2047-00-05

PROPERTY OF EPA REGION 9

HAZARDOUS WASTE MANAGEMENT DIVISION

PLEASE RETURN TO RECORDS CENTER

215 FREMONT STREET

SAN FRANCISCO, CALIFORNIA 94105

EPA WORK ASSIGNMENT NO. 208-9LC1
UNDER
EPA CONTRACT NO. 68-01-7250

FINAL ENDANGERMENT ASSESSMENT

WASTE DISPOSAL, INC.
SANTA FE SPRINGS, CALIFORNIA

NOVEMBER 1989

EPA WORK ASSIGNMENT NO. 208-9LC1
UNDER
EPA CONTRACT NO. 68-01-7250

FINAL ENDANGERMENT ASSESSMENT

WASTE DISPOSAL, INC.
SANTA FE SPRINGS, CALIFORNIA

NOVEMBER 1989

Prepared by:

Clement Associates, Inc.
160 Spear St., Suite 1380
San Francisco, CA 94105

Michael Raybourn
Project Manager
Clement Associates, Inc.

Daniel Melchior
Site Manager
Ebasco Services, Inc.

Dale L. Rowlison
REM III Program Manager
Ebasco Services, Inc.

EPA WORK ASSIGNMENT NO. 208-9LC1
UNDER
EPA CONTRACT NO. 68-01-7250

FINAL ENDANGERMENT ASSESSMENT

WASTE DISPOSAL, INC.
SANTA FE SPRINGS, CALIFORNIA

NOVEMBER 1989

TABLE OF CONTENTS

List of Tables
WDI Endangerment Assessment

List of Figures

EXECUTIVE SUMMARY

The Waste Disposal, Inc. (WDI) site in Santa Fe Springs, California is currently the subject of a Comprehensive Environmental Response Compensation and Liability Act (CERCLA) Remedial Investigation/Feasibility Study (RI/FS) being conducted by the REM III project team under contract to the United States Environmental Protection Agency (EPA).

This endangerment assessment addresses the potential human health and environmental impacts associated with the WDI site under the no-action alternative. An endangerment assessment evaluates potential health risks in the absence of any remedial (corrective) actions. Evaluation of the no-action alternative is required under Section 300.68(f)(v) of the National Contingency Plan. It is based on the site sampling data collected during the remedial investigation (RI) conducted by EPA. The purpose of the endangerment assessment is to provide input for the feasibility study (FS) by identifying the impacts of current levels of contamination on the surrounding environment.

The WDI site consists of several independently-owned parcels of land located in Santa Fe Springs, Los Angeles County, California. The central portion of the site contains a 1,000,000 barrel (42,000,000 gallon) capacity 600-foot diameter, 25-foot deep concrete reservoir and numerous unlined sumps. Adjacent to this central portion of the site are several small businesses. The majority of the site, including the reservoir and the former waste handling areas, is covered with fill materials and a thick growth of vegetation. Most of the land is vacant, but several areas serve as temporary equipment storage sites for local businesses.

Three distinct contaminated areas have been identified within the boundaries of the CERCLA site:

- the reservoir area,
- the eight waste handling areas identified around the reservoir, and

- the former Toxo Spray Dust Inc. site, located west of the reservoir.

The WDI facility began operations in about 1919 for the storage of petroleum from the Santa Fe Springs Oil Field Discovery Area. Between 1949 and 1966, the Waste Disposal, Inc. facility operated as a permitted landfill. From 1956 to 1960, the western portion of the WDI facility operated as an auto wrecking yard.

The exact nature of the wastes accepted at the WDI facility and deposited in the reservoir, the major feature at this site, is not known. Industrial waste permits indicate that rotary drilling mud, clean earth, rock, sand and gravel, paving fragments, concrete, brick, plaster, steel mill slag, and dry mud cake from oil field sumps were disposed of in the reservoir. In 1953, 220 barrels of acetylene sludge were accepted per week. The reservoir was filled with solid material by the end of 1962. Final grading of the site with top soil was completed in 1966.

The second major feature of the WDI site are the eight waste-handling areas. Initially, the waste handling areas were bermed containment ponds created to store wastes when the reservoir reached capacity. Numerous deep sump holes filled with material and oil sludge were present outside the reservoir by 1955, as seen in aerial photographs. From 1957 on, the unlined sumps and the ground surface surrounding the reservoir were used regularly for disposal of liquid wastes. The waste-handling areas not directly adjacent to the reservoir were covered with fill material by 1958.

The third major feature of the WDI site is the Toxo Spray Dust, Inc. (Toxo) area. Toxo began operations by formulating and storing pesticides adjacent to the reservoir in 1953. In 1986, the facility was demolished as required by the California Department of Health Services.

Two off-site releases have been documented (EBASCO, 1988a). The first occurred in 1956 when liquid flowed and was pumped through holes in the dike

around the reservoir into a surrounding channel and then flowed east onto an adjacent property. The second release occurred in 1962 when, after heavy rains, oily liquids seeped through the northern dike onto the St. Paul's High School grounds. During operations at the facility, wastes were also discharged into the Los Angeles County sewer system.

Soil, groundwater, subsurface gas, and air samples were collected from the WDI site and surrounding properties to evaluate the extent of contamination at the site. During the RI, a number of chemicals were identified in environmental media at the site. In accordance with EPA guidance, when a large number of chemicals are detected at a site, a subset of chemicals of potential concern is selected in order to focus the risk assessment on those contaminants that are most likely to pose risks to human health and the environment (EPA, 1986a).

Surface soil samples were collected to a depth of 12 inches. The inorganic compounds selected as chemicals of potential concern for surface soils are antimony, arsenic, cadmium, chromium, copper, lead, mercury, selenium, and thallium. Arsenic, cadmium, and chromium were present at background concentrations, but were selected based on their carcinogenicity. Eight groups of organic chemicals were selected as chemicals of potential concern for soils:

- Chlorinated Pesticides: chlordane, DDT, DDD, and DDE, dieldrin, and heptachlor epoxide;
- Monocyclic Aromatic Hydrocarbons: benzene, ethylbenzene, toluene, and xylenes;
- Organic Acids: benzoic acid;
- Ketones: 2-butanone;
- Polycyclic Aromatic Hydrocarbons: acenaphthene, anthracene, benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene, benzo(a)pyrene, chrysene, dibenzofuran, fluoranthene, fluorene, 2-methylnaphthalene, naphthalene, phenanthrene, and pyrene;
- Chlorinated Aliphatic Compounds: Methylene Chloride;

- Polychlorinated Biphenyls: Aroclor-1248, and Aroclor-1260;
- Phenolic Compounds: Pentachlorophenol.

In addition to evaluating contamination only in the top one-foot of soils, soil samples collected to a depth of 20 feet were also evaluated in this Endangerment Assessment. The subsurface soil contamination was similar to that found in surface soils, with most of the contamination detected between depths of 10 to 20 feet. The chemicals of potential concern selected by chemical class are:

- Inorganics: antimony, arsenic, cadmium, chromium, copper, lead, manganese, mercury, nickel, selenium, thallium, and zinc;
- Chlorinated Aliphatic Hydrocarbons: carbon tetrachloride, chloroform, methylene chloride, tetrachloroethylene, trichloroethylene, and vinyl chloride;
- Chlorinated Pesticides: aldrin, BHC, chlordane, DDT, DDD, DDE, dieldrin, heptachlor, and heptachlor epoxide;
- Monocyclic Aromatic Hydrocarbons: benzene, ethylbenzene, toluene, and xylenes;
- Ketones: 2-butanone;
- Phenolic Compounds: pentachlorophenol;
- Polycyclic Aromatic Hydrocarbons: acenaphthene, acenaphthylene, anthracene, benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene, benzo(a)pyrene, chrysene, dibenzo(a)anthracene, dibenzofuran, fluoranthene, fluorene, indeno(1,2,3-c,d)pyrene, 2-methylnaphthalene, naphthalene, phenanthrene, and pyrene;
- Polychlorinated Biphenyls: Aroclor-1221, Aroclor-1016, Aroclor-1242, Aroclor-1248, Aroclor-1254, Aroclor-1260.

Twenty-seven groundwater monitoring wells were installed and sampled during the RI. In addition, two off-site wells were installed and sampled. Compounds detected at, or downgradient of, the WDI site were compared with the sampling results of two upgradient wells to determine the effect of operations

at the site on the groundwater. Based on a comparison with the upgradient wells and the results of the sampling effort, the following compounds were selected as chemicals of potential concern for groundwater:

- Inorganics: arsenic, lead, manganese, and mercury;
- Chlorinated Aliphatic Hydrocarbons: chloroform, tetrachloroethylene, trichloroethylene;
- Monocyclic Aromatic Hydrocarbons: toluene.

Subsurface gas monitoring wells were installed at 26 locations at the site at depths ranging from 34 to 40 feet. Based on the sampling results, the following compounds were selected as chemicals of potential concern:

- Monocyclic Aromatic Hydrocarbons: benzene;
- Halogenated Aliphatic Hydrocarbons: carbon tetrachloride, chloroform, 1,2-dibromoethane, 1,2-dichloroethane, tetrachloroethylene, 1,1,1-trichloroethane, trichloroethylene, and vinyl chloride.

Although air sampling for airborne particulates was performed, the data were of insufficient quality for EPA data validation criteria. As a result, these data are not presented, or used, in this EA.

The sampling results from the RI were combined with standard exposure assumptions (EPA, 1988) to estimate potential exposures to current and future receptor populations. Pathway-specific estimates of potential exposure point concentrations were combined with information on the toxic potency of the chemicals of potential concern to determine if the WDI site poses a potential risk to human health or the environment.

EPA guidance for conducting quantitative risk assessments directs that concentrations of chemicals at exposure points be compared to applicable or relevant and appropriate (ARARs) regulatory standards and criteria (EPA, 1986a,b,c,d). If regulatory standards are not available for all chemicals in all media, then a quantitative risk assessment must be performed. The

concentrations of chemicals of potential concern in groundwater at the WDI site were compared with available drinking water standards. It was found that lead exceeded the Federal and State of California Maximum Contaminant Level (MCL) and the maximum concentrations of tetrachloroethylene and trichloroethylene exceeded their Federal and State of California MCLs, the Federal MCL for tetrachloroethylene being a proposed standard. Manganese exceeded its secondary Federal and California MCL. The estimated air pathway exposure point concentrations of lead and vinyl chloride were compared with available standards or criteria and found to be below these levels. California Total Threshold Limit Concentrations (TTLCs) are To Be Considered criteria for soils. Available TTLCs were compared to measured soil concentrations. The maximum concentrations of DDT, lead, and zinc in subsurface soils and the maximum concentration of DDT in surface soils exceeded their respective TTLCs.

In order to quantitatively evaluate the potential for endangerment of human health under the current-and future-use exposure scenarios, the estimated chronic daily intake of each chemical for each pathway was compared to critical toxicity values developed by EPA. Reference doses were used as indicators of noncarcinogenic toxicity, and cancer potency factors were used to evaluate carcinogenic risk.

Under current land-use conditions, the principal exposure pathways by which human receptors could potentially be exposed to site contaminants were:

- direct contact with surface soils by trespassers,
- inhalation of airborne particulates and volatiles by residents and students.

The exposure point concentrations of the chemicals of potential concern were estimated for the potentially exposed populations. Human health risks were assessed based on these estimates of exposure and a quantitative description of each compound's toxicity. The major conclusions are summarized in Table ES-1 and below.

Table ES-1
Summary of Potential Health Risks
Waste Disposal, Inc. Site

	Total Upperbound Lifetime Excess Cancer Risks		Noncarcinogenic Hazard Index (CDI:RfD)			
	Average Case	Plausible Maximum Case	Average Case		Plausible Maximum Case	
Current Use						
1. Exposure of Trespassers to surface soils	5×10^{-7}	3×10^{-5}	<1		>1	(3)
2. Exposure of off-site residents to Airborne Particulates						
- 0.1 km	3×10^{-6}	8×10^{-6}	<1		<1	
- 0.5 km	5×10^{-7}	2×10^{-6}	<1		<1	
- 1 km	2×10^{-7}	8×10^{-7}	<1		<1	
3. Exposure of Students to Airborne Particulates	2×10^{-7}	4×10^{-7}	<1		<1	
4. Exposure of off-site Residents to Airborne Volatile Chemicals						
- 0.1 km	3×10^{-7}	5×10^{-6}	<1		<1	
- 0.5 km	5×10^{-8}	1×10^{-6}	<1		<1	
- 1 km	2×10^{-8}	5×10^{-7}	<1		<1	
5. Exposure of Students to Airborne Volatile Chemicals	3×10^{-8}	3×10^{-7}	<1		<1	
Future Use						
1. Exposure of On-site Residents to Surface Soils						
- Adults	3×10^{-6}	7×10^{-4}	<1		>1	(10)
- Children (1-6 years)	2×10^{-5}	3×10^{-3}	>1	(2)	>1	(500)
2. Exposure of On-site Residents to Groundwater						
- Adults	4×10^{-5}	3×10^{-4}	<1		>1	(2)
- Children			>1	(2)	>1	(8)
3. Exposure of On-site Residents to Volatiles in Indoor Air						
- Adults	6×10^{-5}	6×10^{-4}	<1		<1	
- Children			<1		<1	

The results of the risk evaluation indicate that under current-use conditions, direct contact with soils by trespassers and inhalation of airborne particulates by off-site residents pose the greatest public health risk. If the land use conditions at the WDI site were to change to residential use, direct contact with surface soils by on-site residents and inhalation of subsurface gas contaminants in indoor air would pose the greatest public health risks. These values are not adjusted to eliminate contributions to risk from background levels of the chemicals of concern or to incorporate other source of chemical intake such as occupational exposure.

Exposure of potential trespassers on the WDI site to contaminated surface soils resulted in potential carcinogenic risks of 5×10^{-7} (five-in-ten million) under average conditions and 3×10^{-5} (three-in-100 thousand) under plausible maximum conditions. Exposure under average conditions to the noncarcinogens evaluated under this exposure scenario are not expected to result in adverse health effects since the Hazard Index (the sum of the CDI:RfD ratios) for average exposures was less than one. For the plausible maximum case, the Hazard Index was greater than one, primarily due to the CDI:RfD ratio for lead which was equal to one.

Exposures to airborne dust particles was predicted by mathematical modeling of the dispersion of fugitive dust moving from surface soils into the air. Inhalation of airborne particulates by residents living 0.1 km from the site could result in 3×10^{-6} and 8×10^{-6} potential upperbound excess lifetime cancer risks for the average and plausible maximum exposure cases, respectively. Potential carcinogenic risks of 5×10^{-7} for the average exposure case and 2×10^{-6} for the plausible maximum case were calculated for residents living 0.5 km from the site. Under average exposure conditions, residents living 1 km from the site may experience a potential upperbound carcinogenic risk of 3×10^{-7} and, under plausible maximum conditions, a 8×10^{-7} potential upperbound carcinogenic risk. Since the Hazard Indices are all less than one, exposure to the chemicals exhibiting noncarcinogenic effects appears to present a low probability of adverse health effects under

both average and plausible maximum exposure at all three distances from the site.

If dust generation were to occur during remediation of the site, the risks associated with inhalation of fugitive dust may be higher since greater amounts of soil may be exposed. The FS should consider risks associated with airborne dust exposure during remedial activities and present dust suppression measures.

Residents living near the WDI site could also be exposed to volatile organic compounds (VOCs) volatilized from soils at the site. Inhalation of these VOCs by residents living 0.1 km from the site could result in estimated 3×10^{-7} and 5×10^{-6} potential upperbound excess lifetime cancer risks for the average and plausible maximum exposure cases, respectively. Potential carcinogenic risks were estimated to be 5×10^{-8} for the average case and 1×10^{-6} for the plausible maximum case for residents living 0.5 km from the site. Under average exposure conditions, residents living 1 km from the site might experience a potential upperbound carcinogenic risk of 2×10^{-8} and, under plausible maximum conditions, a 5×10^{-7} potential upperbound carcinogenic risk. Exposure to the chemicals exhibiting noncarcinogenic effects appears to present a low probability of adverse health effects based on the conditions of both average and plausible maximum exposure at all three distances from the site as the Hazard Indices are all less than one.

Students attending St. Paul's High School adjacent to the site could inhale airborne particulates or volatile chemicals released from the site. The calculated potential upperbound carcinogenic risks from inhalation of airborne particulates are 2×10^{-7} for the average exposure case and 4×10^{-7} for the plausible maximum case. The calculated average and plausible maximum potential upperbound carcinogenic risks from inhalation of volatile organic chemicals are 3×10^{-8} and 3×10^{-7}, respectively. Exposure to the chemicals exhibiting noncarcinogenic effects appears to present a low probability of adverse health effects based on the conditions of both average and plausible

maximum exposure to both sources of inhalation exposure, as the Hazard Indices are all less than one.

The exposure scenarios described above would also apply for future land-use conditions. In addition, exposure pathways were evaluated for direct contact with on-site surface soils by residents of two separate age groups, ingestion of groundwater when used as a primary drinking water source by adults and children, and inhalation of volatiles inside dwellings on the site. The results of the risk assessment are also presented in Table ES-1 and are summarized below.

If the WDI site were to be developed for residential purposes, residents could be exposed to contaminants present in the surface soils. Under this future use scenario, soils that are currently at depths of up to 20 feet could become surface soils as a result of excavation during development. Risks for two age groups were quantified: adults and young children aged 1 to 6 years. The potential upperbound excess cancer risks for adults were 3×10^{-6} under average exposure conditions and 7×10^{-4} under plausible maximum exposure conditions. Exposure to the noncarcinogenic compounds would result in a Hazard Index below one under average conditions and a Hazard Index greater than one under plausible maximum conditions. For young children, the potential upperbound excess cancer risk was estimated to be 2×10^{-5} under average conditions and 3×10^{-4} under plausible maximum conditions. The potential for human health risk from noncarcinogenic chemicals does exist since the Hazard Indices for both the average and plausible maximum cases were greater than one. Under average conditions, the CDI:RfD ratio for thallium exceeded one. Under plausible maximum conditions, the CDI:RfD ratios for antimony, arsenic, cadmium, chromium, copper, lead, manganese, thallium, zinc, chlordane, and DDT all exceeded one.

The groundwater beneath the WDI site could potentially be used as a drinking water source. Exposure to individuals ingesting this water and using it for residential purposes was evaluated. Under average exposure conditions, the potential upperbound excess cancer risk was estimated to be 4×10^{-5},

while under plausible maximum conditions, the potential upperbound excess cancer risk was 3×10^{-4}. Under average exposure conditions, the Hazard Index is less than one, while under plausible maximum exposure, the Hazard Index is greater than one, due primarily to the presence of arsenic, lead, and manganese. Noncarcinogenic risks for small children consuming groundwater underlying the WDI site may pose a health threat since the Hazard Indices for both average and plausible maximum cases exceed one, due primarily to the presence of arsenic, lead, and manganese.

The volatile organic chemicals of potential concern present in soils could migrate vertically and enter buildings through their foundations if the site were developed for residential purposes. Exposure to potential future on-site residents to indoor air were estimated to result in potential upperbound excess cancer risks of 6×10^{-5} and 6×10^{-4} under average and plausible maximum exposure conditions, respectively. Exposure to the chemicals exhibiting noncarcinogenic effects appears to present a low probability of adverse health effects for both children and adults, based on both average and plausible maximum exposures to both sources of inhalation exposure, as the Hazard Indices are all less than one. In addition, it does not appear that the gas is reaching the surface in appreciable concentrations based on field measurements.

A considerable amount of uncertainty is associated with the assessment of risks posed by a particular site. In making exposure assumptions and selecting modeling parameters, an attempt was made to use assumptions that are unlikely to underestimate risk.

The environmental assessment qualitatively evaluated potential impacts to biotic receptors associated with the chemicals of potential concern at the WDI. The effects of the inorganic chemicals of potential concern on wildlife are not well documented. Federally endangered species potentially present at the site include the island night lizard, slender-horned spineflower, American peregrine falcon, and Least Bell's vireo. The adverse impact of pesticides on

birds of prey and other wildlife has been documented. A potential present or future threat to wildlife at the site may exist.

1.0 INTRODUCTION

This report presents a quantitative endangerment assessment for the Waste Disposal, Inc. (WDI) site located in Santa Fe Springs, California. An endangerment assessment (EA) is prepared as part of the evaluation of a site subject to clean-up action under the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA) of 1980, as amended in 1986 under the Superfund Amendments and Reauthorization Act (SARA). An endangerment assessment is performed to evaluate the impact of the no-action remedial alternative and to assess if an actual or threatened release of a hazardous substance from the site may present an imminent or substantial endangerment to public health or welfare or the environment.

This assessment follows EPA guidance for risk assessment in general and or Superfund sites in particular (EPA, 1986a,b,c,d; EPA, 1988e,f) and is based mainly on data generated during the Remedial Investigation (RI) conducted by EBASCO (1988a,b; 1989a,b,c,d). This EA has been conducted using generally conservative assumptions according to the EPA general guidelines. The purpose of using conservative assumptions is to explore the potential for adverse health effects using conditions that tend to overestimate risk so that the final estimates will be near or higher than the upper end of the range of actual exposures and risks. As a result, this risk assessment should not be construed as presenting an absolute estimation of risk to human or environmental populations; rather, it is a conservative analysis intended to indicate the potential for adverse impact to occur.

In Section 2 of this endangerment assessment, the WDI site is described and the history of the site is summarized, the nature and extent of chemical contamination at the site is characterized, and the chemicals considered most likely to pose a health risk (the contaminants of potential concern) are identified. Section 3 summarizes the toxic properties of the contaminants from a human health standpoint. Section 4 presents information on the environmental fate and transport of the chemicals of concern, evaluates potential exposure pathways, and calculates contaminant concentrations at

potential receptors. An evaluation of potential risks to individuals living in the vicinity of the WDI site is presented in Section 5 along with a discussion of the uncertainties in the assessment; a qualitative ecological assessment is also presented. Appendix A presents detailed toxicity profiles for those contaminants of concern that present the greatest risks under the scenarios considered in this assessment. Details of the estimation methods used to predict exposure point concentrations are discussed in Appendix B. Appendix C provides sample calculations of the risk estimation techniques used in Section 5.

2.0 SITE CHARACTERIZATION

Characteristics of the WDI site will influence the manner in which individuals may be exposed to contaminants. Historical, physical, environmental, and chemical characteristics of the WDI site are summarized below and discussed in detail in the Remedial Investigation (RI) Reports prepared by EBASCO Services, Inc. for EPA (EBASCO, 1988a,b; 1989a,b,c).

2.1 SITE DESCRIPTION AND HISTORY

The WDI site consists of several individually-owned parcels of land located in Santa Fe Springs, Los Angeles County, California (see Figure 2-1). The 43-acre site is bordered on the northwest by Santa Fe Springs Road, on the northeast by Fedco food distribution center and St. Paul's High School's athletic field and parking lot, on the southwest by Los Nietos Road, and on the southeast by Greenleaf Avenue. The central portion of the site contains a 1,000,000 barrel capacity (42,000,000 gallon) concrete reservoir and numerous unlined sumps. The reservoir is 600 feet in diameter and 25 feet deep. These are shown in Figure 2-2. Adjacent to this central area are several parcels of land on which a variety of small businesses are currently located. The majority of the site is covered with fill materials and a thick growth of vegetation. The central portion of the site is approximately 10 to 20 feet above the surrounding terrain. Steep dropoffs are located along the northern and western borders of the site. Most of the land is vacant but several areas serve as temporary equipment storage sites for local businesses. Local residents and workers appear to be using this portion of the site to access surrounding properties.

The preliminary boundaries of the WDI site, as given in post-1949 Los Angeles County permits, identified the central reservoir and sump portion as the area of immediate concern. However, after EPA reviewed historical information about the area and historical aerial photographs, the boundaries were extended to include the entire area used for landfilling operations. The

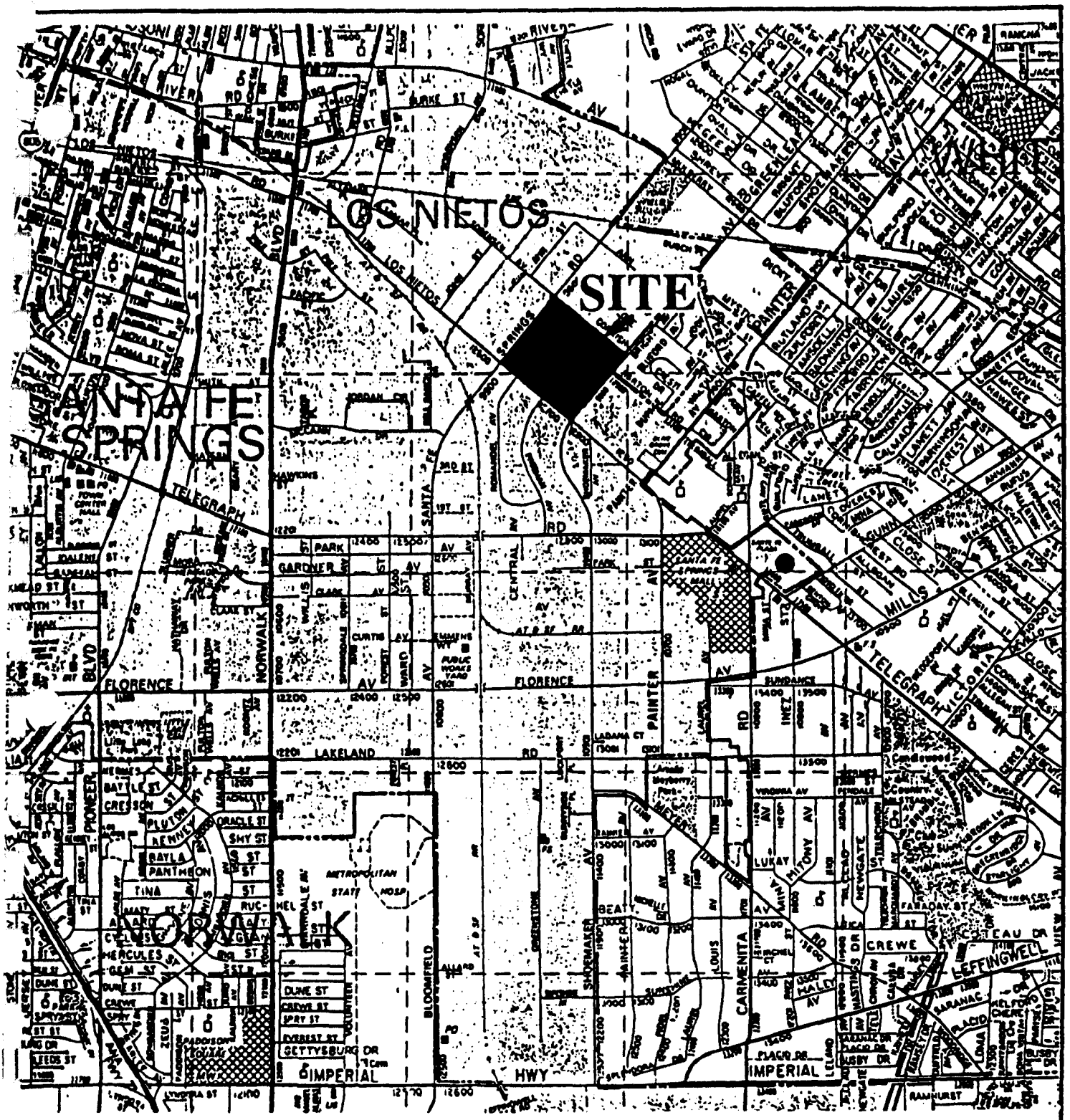
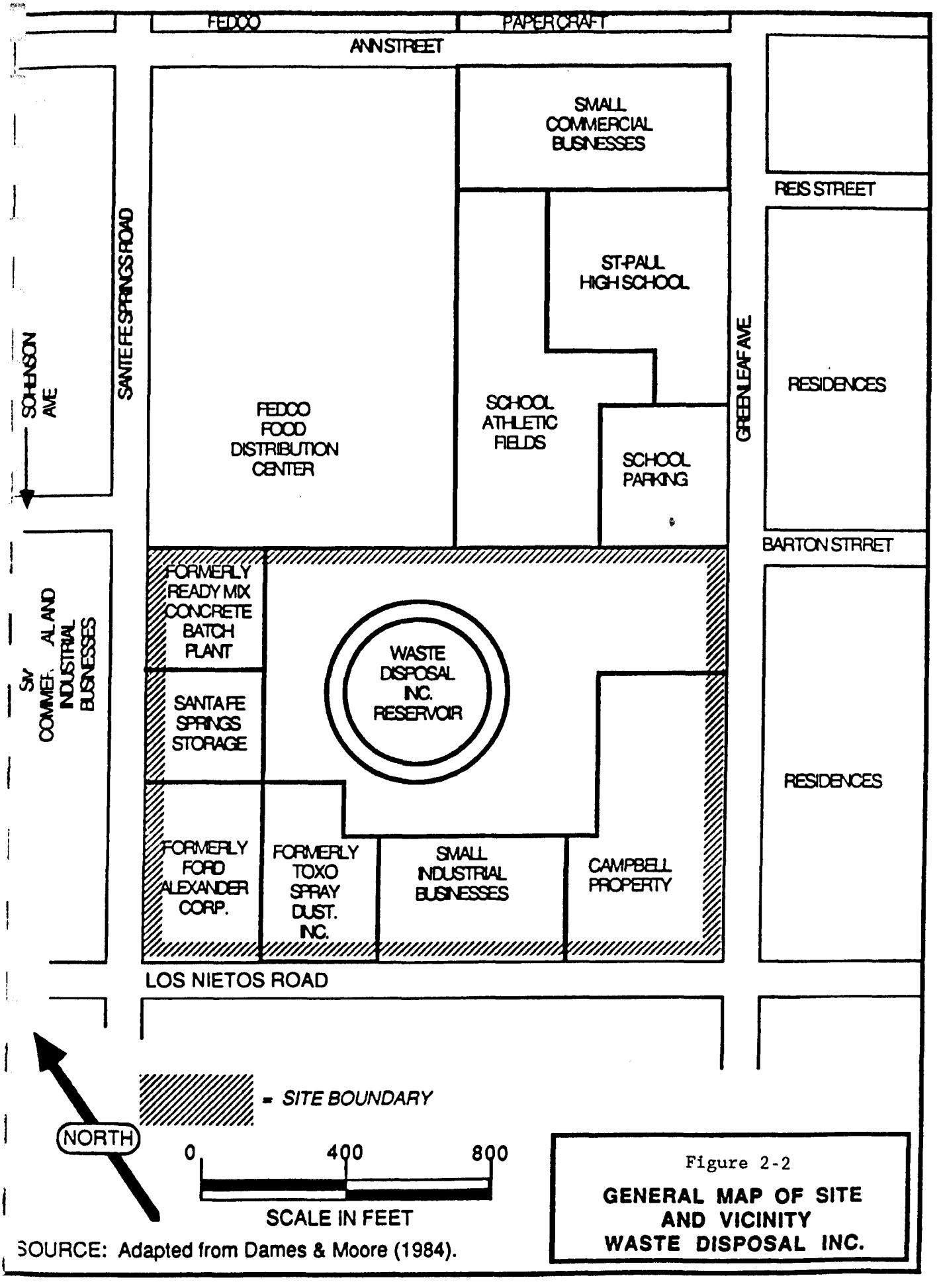


Figure 2-1
GENERAL SITE LOCATION MAP
Waste Disposal Inc.

SOURCE: Adapted from Thomas Brothers Guide, 1988.



SOURCE: Adapted from Dames & Moore (1984).

Figure 2-2

GENERAL MAP OF SITE AND VICINITY WASTE DISPOSAL INC.

current site is believed to encompass the maximum probable area of suspected contamination. Three areas have been identified within these boundaries as possible sources of contamination. These areas include: 1) the former WDI facility, 2) the eight sumps identified around the reservoir, and 3) the former Toxo Spray Dust Inc. (Toxo) site, west of the reservoir.

2.1.1 Reservoir Area

The reservoir area began operations in about 1919 for the purpose of storage of petroleum from the Santa Fe Springs Oil Field discovery area. The concrete petroleum storage reservoir is believed to have been constructed some time between 1919 and 1928. It was decommissioned as a petroleum storage facility in the late 1920s. The initial structure was surrounded on three sides by an earthen dike surrounded by a drainage channel. Early operations at the facility were unregulated. Between 1949 and 1965-66, the WDI facility operated as a permitted landfill. From 1956 to 1960, an auto wrecking yard operated on the western portion of the WDI site.

In 1949, a permit was granted by the County of Los Angeles Board of Supervisors for the disposal of solid fill, rotary mud, and other non-acid oil well wastes at the facility (EBASCO, 1989a). Information was not available regarding any materials remaining in the reservoir prior to when it was permitted in 1949. In 1950, the County of Los Angeles issued an Industrial Waste Permit to WDI allowing the acceptance of rotary drilling mud, clean earth, rock, sand and gravel, paving fragments, concrete, brick, plaster, steel mill slag, and dry mud cake from oil field sumps. In early 1953, the facility began operating 24-hours per day. Later in 1953, the Department of County Engineers in Los Angeles permitted the acceptance of 220 barrels of acetylene sludge per week from Security Engineering and Chicksan Company (EBASCO, 1989a). Disposal of drilling muds was moved to an area west of the reservoir that was annexed by WDI in 1955 (O'Grady, 1955; Pitts, 1955; Breivogel, 1955, 1956). Beginning in 1958, solid fill was accepted and used to grade over the site (Granich, 1958). By the end of 1962, the reservoir was

completely full of solid material (Moore, 1962). Final grading of the site with top soil continued until 1966 when the landfill was closed. The site was closed to the public in 1964.

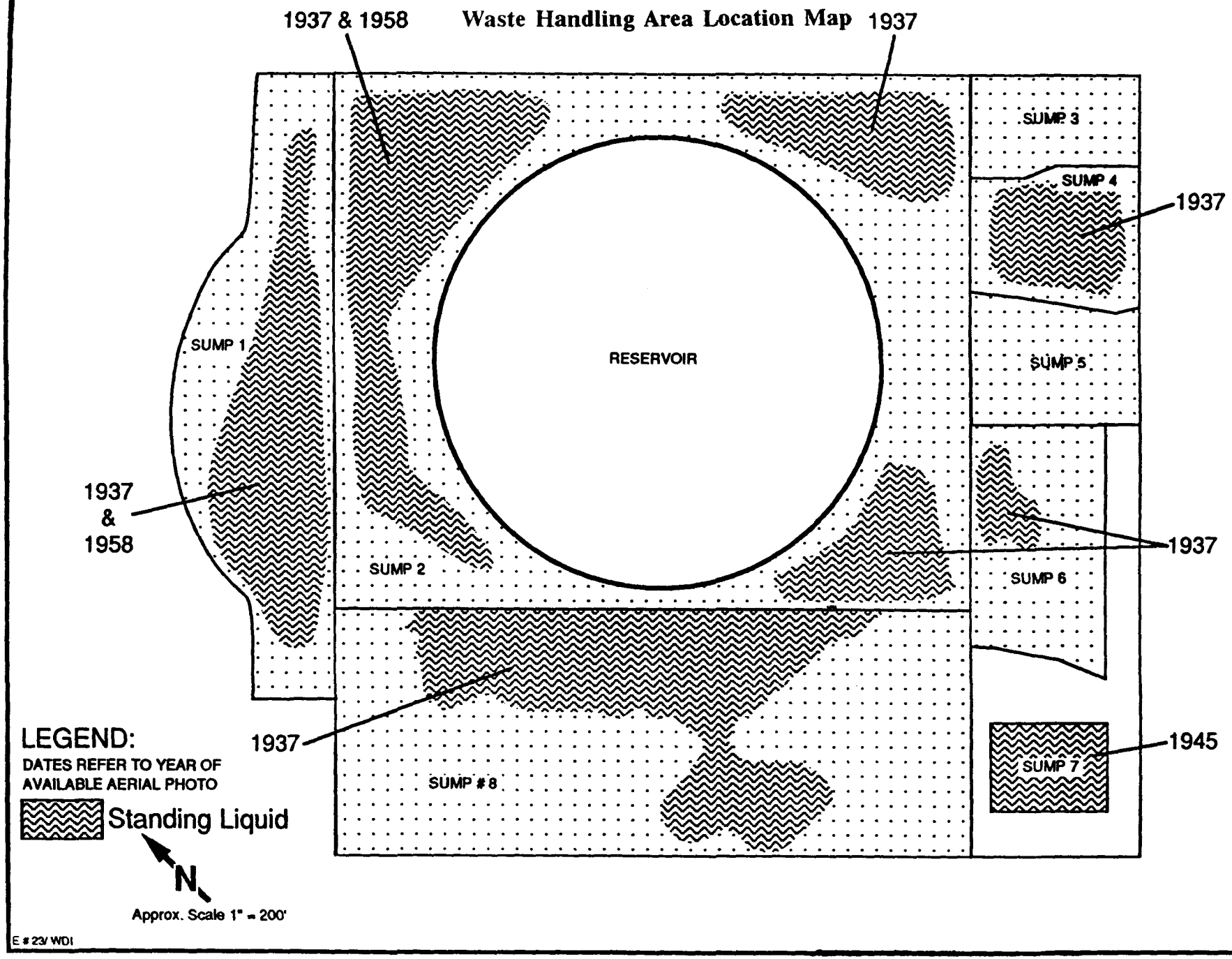
2.1.2 Waste Handling Areas

The unlined waste handling areas surrounding the WDI reservoir are the second feature area identified as a potential source of contamination. Aerial photographs, drilling logs, and cone penetrometer tests were used to locate a total of eight waste handling areas on the WDI site (see Figure 2-3). The true extent of the waste handling areas is speculative at this time, as aerial photos have shown that the waste handling areas were larger than could actually be sampled under the planned scope of the RI (EBASCO, 1989a); therefore, potential sources of contamination may not have been samples. Initially, the waste handling areas were bermed containment ponds created to store wastes when the reservoir reached capacity. Based on soil boring logs and geophysical surveys of the site, many of the waste handling areas appear to be underlain by indigenous clays and sands. By 1955, the reported presence of numerous deep sump holes filled with material and oil sludge occupying many areas outside the reservoir had been reported (Tweedy, 1950; WDI, 1955). In 1957, an employee was observed pumping liquid from the reservoir to an adjacent unlined waste handling area (Otteson, 1957). After this incident, the unlined waste handling areas as well as the ground surface surrounding the reservoir were used regularly for the disposal of liquid wastes (EBASCO, 1989b). By 1958, the waste handling areas which were not directly adjacent to the reservoir were covered with fill material. This fill material has mixed with the disposed materials to stabilize the liquid materials.

2.1.3 Toxo Spray Dust

Toxo Spray Dust, Inc. (Toxo) is the third feature area identified as a potential source of contamination. Toxo began operations adjacent to the WDI

Figure 2-3


1937 & 1958
Waste Handling Area Location Map
1937
SUMP 3
SUMP 4
1937
SUMP 1
RESERVOIR
SUMP 5
1937
&
1958
SUMP 2
1937
SUMP 6
1937
SUMP # 8
SUMP 7
1945
LEGEND:
DATES REFER TO YEAR OF AVAILABLE AERIAL PHOTO
Standing Liquid
N
Approx. Scale 1" = 200'
E # 23/ WDI

reservoir in 1953, according to aerial photos taken at that time. Pesticide formulation and storage were the main operations of this facility. Closing of this facility was not mentioned in any of the RI reports reviewed. Soil samples, taken in this area in 1986 by Dames & Moore contained a variety of pesticides, including both organochlorine and organophosphate insecticides (Dames and Moore, 1986a,b). As a result of this sampling effort, the California Department of Health Services required demolition of the building in 1986; the remains were hauled to a permitted hazardous waste landfill for disposal (EBASCO, 1988a).

2.1.4 Waste Disposal Practices

Waste stream releases and disposal practices have been infrequently documented for the WDI facility. During WDI's operation, only two off-site releases were documented. The first, in 1956, reported that liquid flowed and was pumped through holes in the dike into a surrounding channel, and then east toward Greenleaf Road and onto adjacent property (EBASCO, 1989b). The second release occurred in 1962 when, after heavy rains, oily liquids seeped through the northern dike onto the St. Paul's High School grounds (EBASCO, 1989c).

When disposal of liquids in the reservoir was discontinued in November, 1962, liquid wastes were occasionally disposed of on the ground. Employees of B & H Vacuum and Hollbrook and Sons were both observed dumping liquids directly onto the ground between 1958 and 1965 (EBASCO, 1989a). "Ponding" of these liquid wastes also occurred along the WDI entrance road from Los Nietos Road (EBASCO, 1989c).

WDI wastes were also discharged to the Los Angeles County sewer system. It appears that waste water was discharged into a channel leading to Greenleaf Avenue. Some time later, a pipe was installed so that liquids could flow directly to Greenleaf Avenue and into the sanitary sewer. With the approval of the Los Angeles Department of Sanitation in 1960, a pipe from WDI was connected to the Los Angeles County sewer system (EBASCO, 1989c). Throughout

its operation, WDI was the cause of numerous complaints from area residents; common complaints included excessive noise, odor, and dust (EBASCO, 1988b).

2.2 SURROUNDING AREA

Presented below is a discussion of the area surrounding the WDI site, including land-use and demographic information. This information will be used to identify potential receptors and sensitive populations for the site.

2.2.1 Land Use

Figure 2-2 depicts the current land use at the WDI site, which is zoned primarily as light industrial and commercial. Local residences border the site to the east and southeast, on Greenleaf Avenue. St. Paul's High School is located just northeast of the site. Many small businesses are located within the boundaries of the site. To the north and west of the WDI site is a food distribution center consisting of one large warehouse, a water tank, and storage yard. Within the site boundaries, west of the reservoir, is a large building housing several companies including Steel Craft Boats, F & H Garcia Plumbing, Xerographics, B & L Engineering, J & M Custom Wood Products, Label Mill, and K-K Chemtronics. On the northwestern side of the WDI site is a large building housing Mersits' Equipment Sales and Service. Also within the site boundaries is Santa Fe Springs Storage located between Mersits' and Steel Craft Boats and consist of a trailer office and a large asphalt-covered lot where recreational vehicles are stored. A narrow access road connects the RV storage facility with Santa Fe Springs Road. An oil well logging company (Dia-log) lies to the west (EBASCO, 1989c).

2.2.2 Demographics

Approximately 70,000 people live within a 2-mile radius of the WDI site (CACI, 1989). Potential sensitive populations, as defined by EPA (1986a), of young and elderly individuals, make up approximately 20 percent of the residential population, including 9% under 4 years old and 10.4% greater than 65 years old. The largest age group (23%) of the current population is between 22 and 29 years. The current growth rate for the area is 0.2% with the projected age distribution through 1994 remaining constant. The average age for this area is 33 years (CACI, 1989). The current student population at St. Pauls's High School is approximately 1,220.

2.3 ENVIRONMENTAL CHARACTERISTICS

The potential impacts of contaminants at any site partially depend on the environmental characteristics of the site and surrounding area. Contaminant migration is influenced by local meteorology, topography and surface drainage, geology, and hydrogeology. These environmental characteristics of the WDI site are described briefly below.

2.3.1 Meteorology

The mean annual precipitation of the site varies from 17 to 20 inches with nearly all of the rainfall occurring during the months of December through March (EBASCO, 1989c). Significant precipitation during the summer months is infrequent, and rainless periods of several months are common. Mean annual evapotranspiration is approximately 52 inches. A meteorological station approximately 10 miles upwind of the WDI site has reported an annual average wind speed of 8.6 meters/sec with the wind predominantly blowing from the southwest (EBASCO, 1988a). Another meteorological station, which was located near the center of the site during the RI, reported an average wind velocity of 5.3 miles/hour (2.6 m/s) from August 24 to September 23, 1988,

also with the wind predominantly from the southwest (EBASCO, 1989d). Figure 2-4 presents a wind rose for the National Weather Service's meteorological station in Rivera; these wind conditions are believe to be indicative of the WDI site (EBASCO, 1989f). As indicated by the wind rose, the wind blows from the south-southwest quadrant approximately 38 percent of the time, from the north-northeast quadrant 27.5 percent, from the south-southeast quadrant 22.5 percent overall, and from the north-northwest quadrant approximately 12 percent overall.

2.3.2 Topography and Surface Drainage

The surface elevation of the WDI site varies from 150 to 168 feet above mean sea level. The central portion of the site overlying the reservoir has the highest elevation; this area is situated 10 to 20 feet above the surrounding terrain. Land to the west and southwest of the reservoir is fairly flat, but steep slopes are present at several other locations on the site. Steep dropoffs occur along the northern border with St. Paul's High School and the food distribution center (30 to 50 percent slope) and along the western edge of the site between the Santa Fe Springs recreational vehicle storage lot and Sleek Craft Boats (45 percent slope). The drop in elevation to the east and southeast is more gradual (EBASCO, 1989b).

Surface drainage on the WDI site is complicated due to slope variations and depressions in the site surface due to prior filling operations. The central portion of the site contains many minor ridges and depressions and water has a tendency to pond in this area. The steep northern and eastern slopes drain the nearby areas and general drainage for the southern half of the site is to the south and east (EBASCO, 1989c). The topographical features of the WDI site are indicated in Figure 2-5.

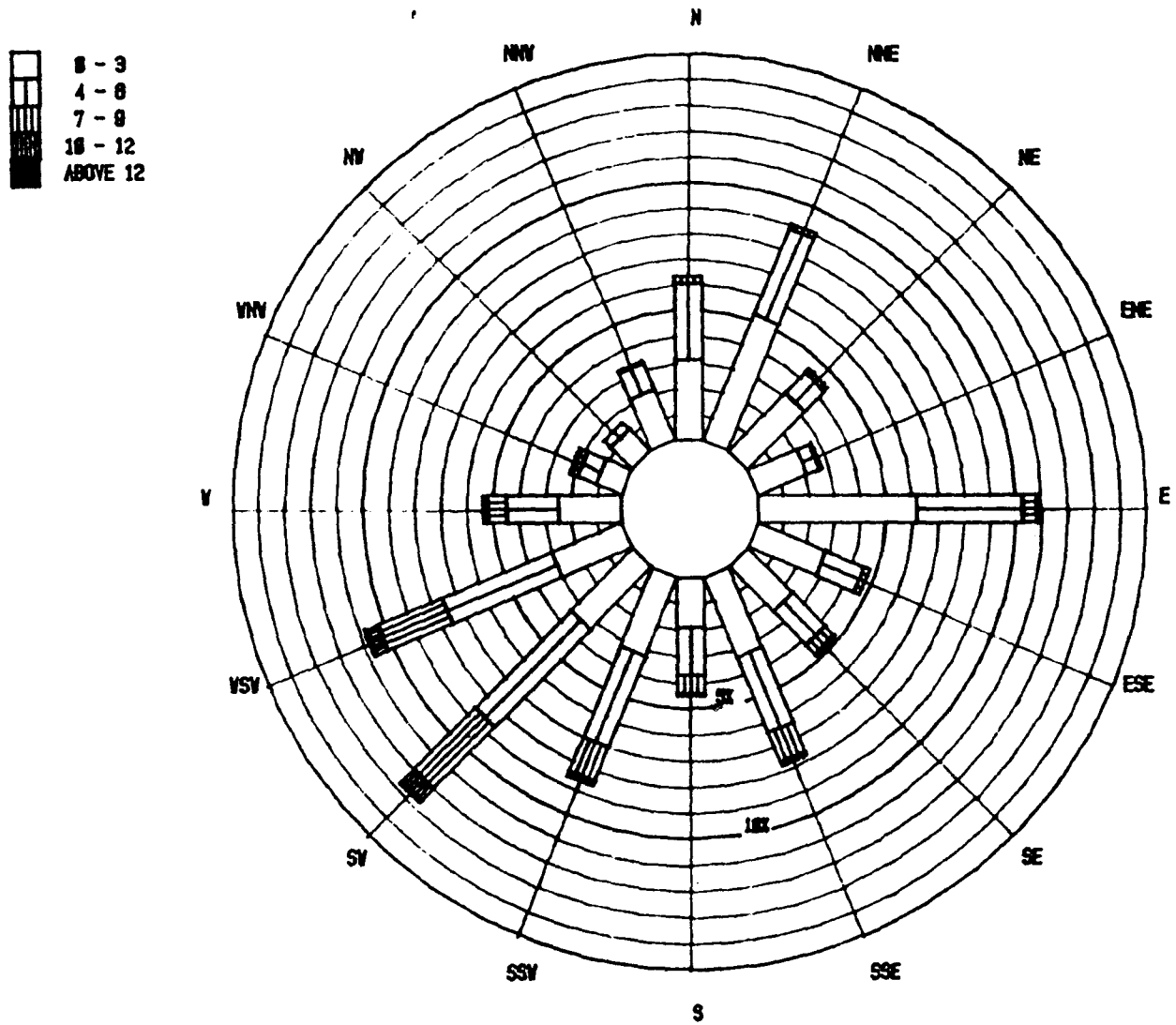


Figure 2-4 WIND ROSE AT RIVERA (1957-1975)

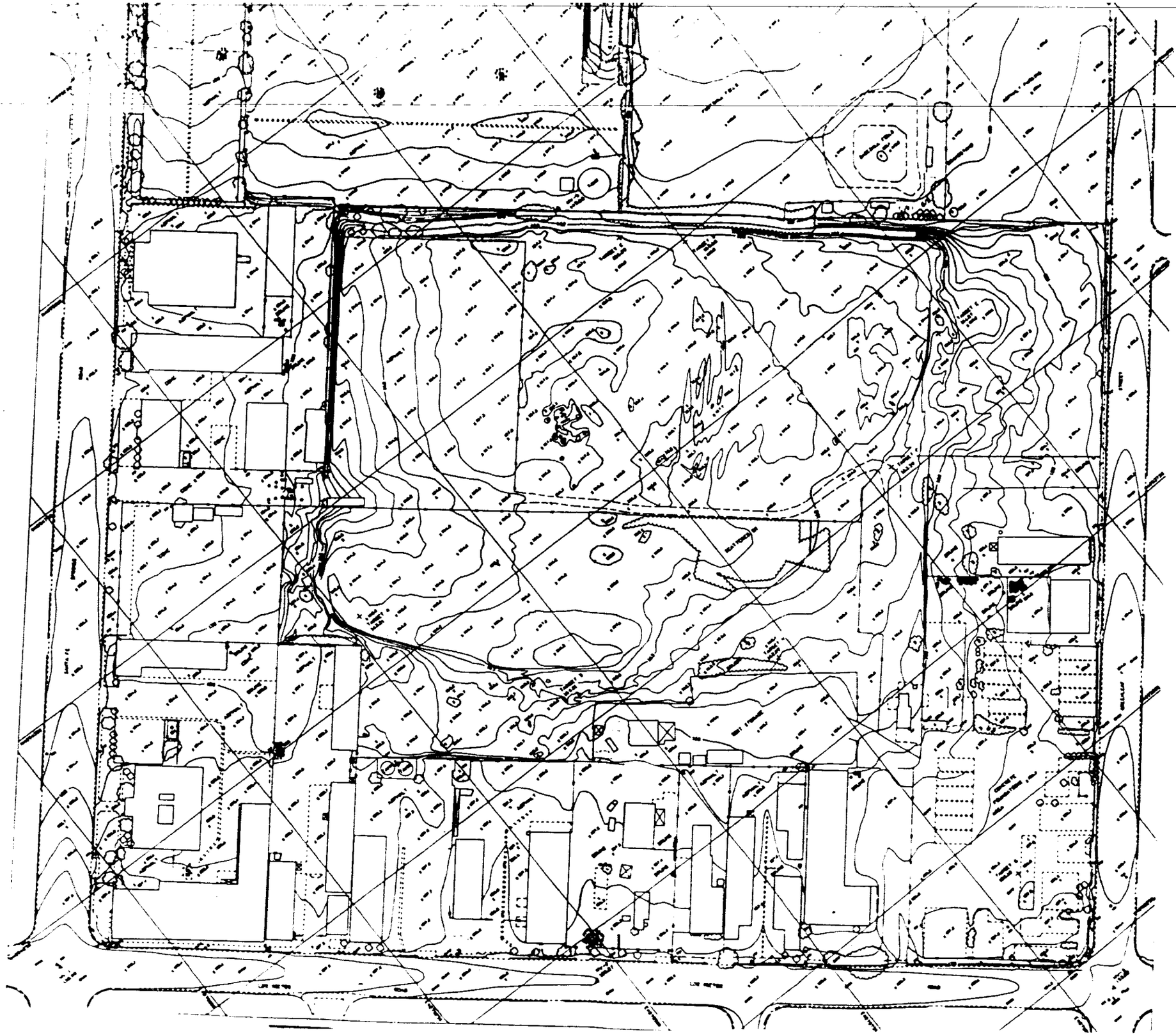


Figure 2-5
TOPOGRAPHIC MAP OF WASTE DISPOSAL INC.

SCALE 1" = 150'

2.3.3 Geology

The WDI site is located northwest of the Santa Ana Mountains, a dominant part of the northern peninsular ranges of coastal southern California and the one which forms the eastern margin of the Los Angeles Basin (Figure 2-6). Situated in the central block of the Los Angeles Basin, the WDI site is bounded on the northeast by the La Habra syncline and on the southwest by the Coyote Hills (Santa Fe Springs) anticline in an area commonly referred to as the Santa Fe Springs Plain. This plain is a gently rolling topographic feature which has probably been warped by the Santa Fe Springs-Coyote Hills anticlinal system and dips gently both to the northeast toward Whittier and to the southeast toward the Downey Plain. The difference in elevation ranges from 100 to 175 feet above sea level (DWR, 1961). The surface of the Santa Fe Springs Plain and the Coyote Hills reflects a structural high which trends northwest from the Coyote Hills in Orange County and is primarily developed in underlying formations of Miocene and Pliocene age. In these sediments, the uplift consists of anticlinal folds which contain the Santa Fe Springs, Leffingwell, and West Coyote oil fields (EBASCO, 1989a).

The general geologic characteristics (stratigraphy) beneath the WDI site have also been examined. Five to fifteen feet of artificial fill material covers the entire site. Below the fill material is a silt layer ranging from 10 to 25 feet in thickness and below the silt layer is a sandy, pebbly, channelized network of braided river deposits at least 50 feet thick. A clay and silt layer about 10 feet thick and from 30 to 40 feet below ground level is predominantly present under the southeast end of the site. This layer is interbedded with the braided river deposits. The apparent direction of sediment transport is in a northeast-southwest direction (EBASCO, 1989b). Figure 2-7 depicts a generalized, composite, northwest-southeast tending cross section of the WDI site.

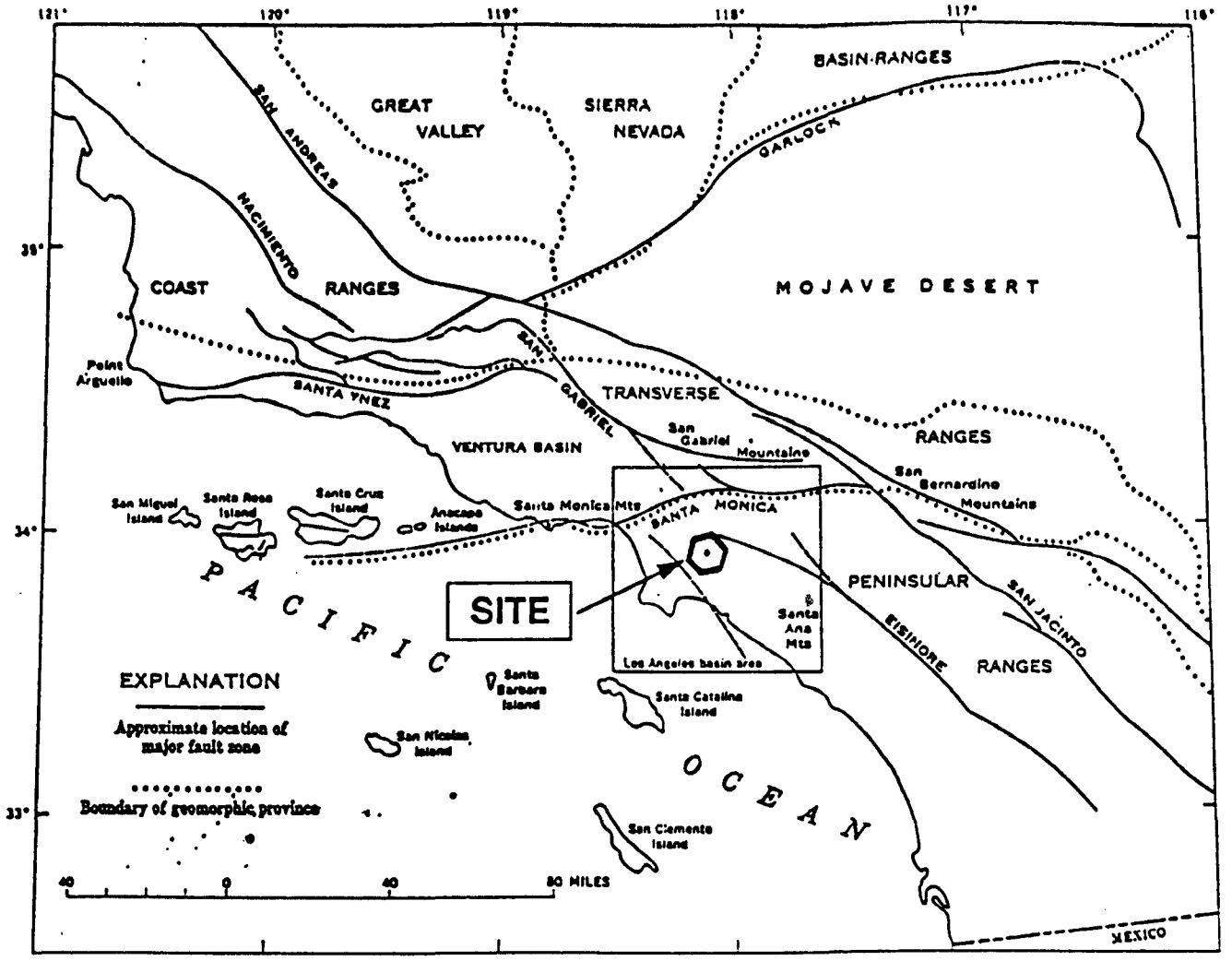


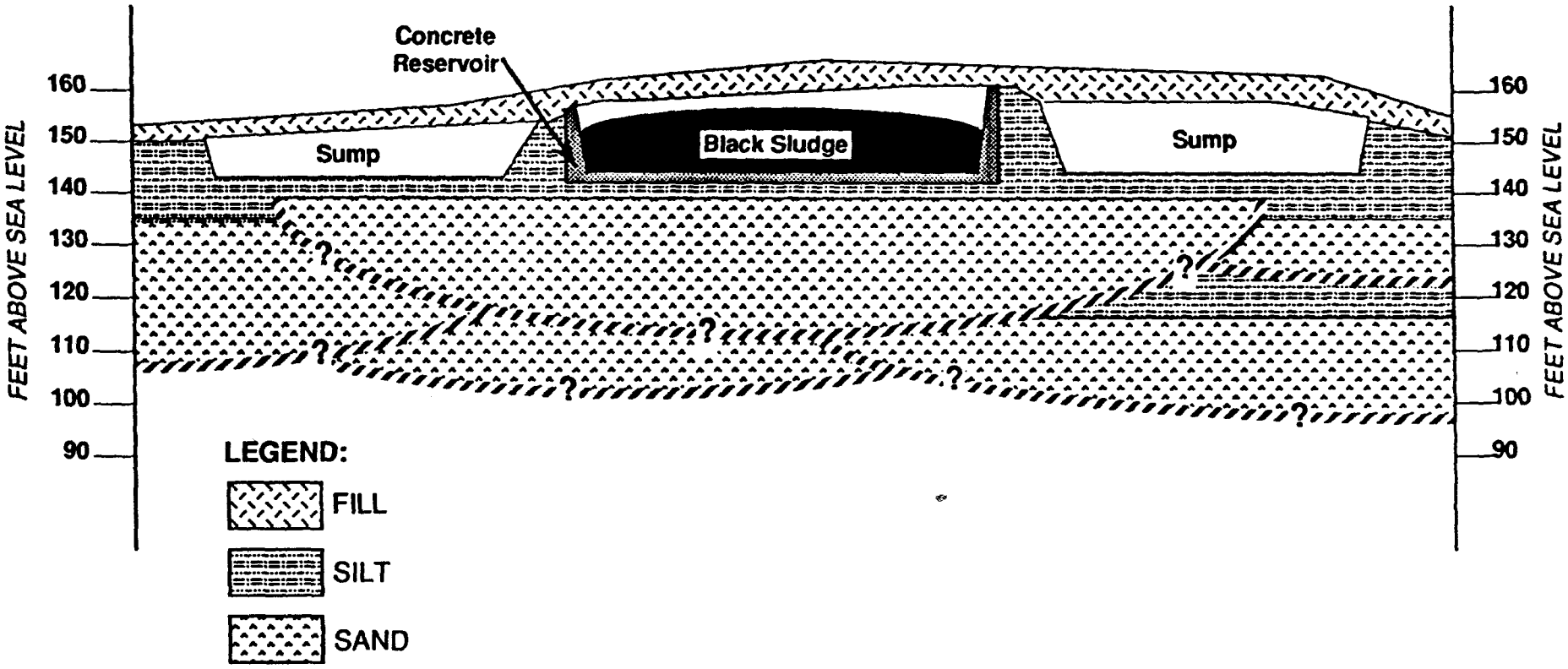
Figure 2-6

GEOMORPHIC PROVINCES AND MAJOR FAULT ZONES

SOURCE: Adapted from Yerkes, R.F., et. al. (1965).

Figure 2-7

GENERALIZED CROSS SECTION ACROSS WDI SITE



HORIZONTAL SCALE:
Approx. 1" = 200'

2.3.4 Hydrogeology

Hydrogeologic characteristics of the region surrounding the WDI site and of the WDI site will influence how chemicals are released from the site. Discussed below are the regional and site specific hydrogeologic conditions for the WDI site.

REGIONAL HYDROGEOLOGY

The WDI site is situated in the Whittier Area of the Central Groundwater Basin. The Whittier Area extends from the Puente Hills south and southwest to the axis of the Santa Fe Springs-Coyote Hills uplift. The western boundary is an arbitrary line separating the Whittier Area from the Montebello Forebay Area; the eastern boundary is the Los Angeles-Orange County line (EBASCO, 1989c). Figure 2-8 displays all of the regional groundwater basins. Figure 2-9 presents a cross-sectional view of the Whittier Area.

The Whittier Area is overlain by the La Habra Piedmont Slope and part of the Santa Fe Springs Plain and the Coyote Hills. The known water-bearing sediments, extending to a depth of about 1,000 feet (800 feet below sea level), include recent alluvium and the Lakewood and San Pedro Formations. A part of the underlying Pliocene and older deposits may also contain water of good quality.

Recent alluvium in the Whittier Area consists of a thin layer of sand, gravel, and clay, which extends into the western portion of the area from the Montebello Forebay Area. The sediments are 80 feet thick near the western boundary of the area, and thin out to the east. The recent alluvium contains a portion of the Bellflower aquiclude, another water bearing zone.

The Bellflower aquiclude in the recent alluvium consists of clay and sandy clay ranging from 10 to greater than 40 feet in thickness. Beneath the Santa Fe Springs Plain, the Bellflower aquiclude is part of the undifferentiated Lakewood Formation. Lack of data from the areas where the Lakewood

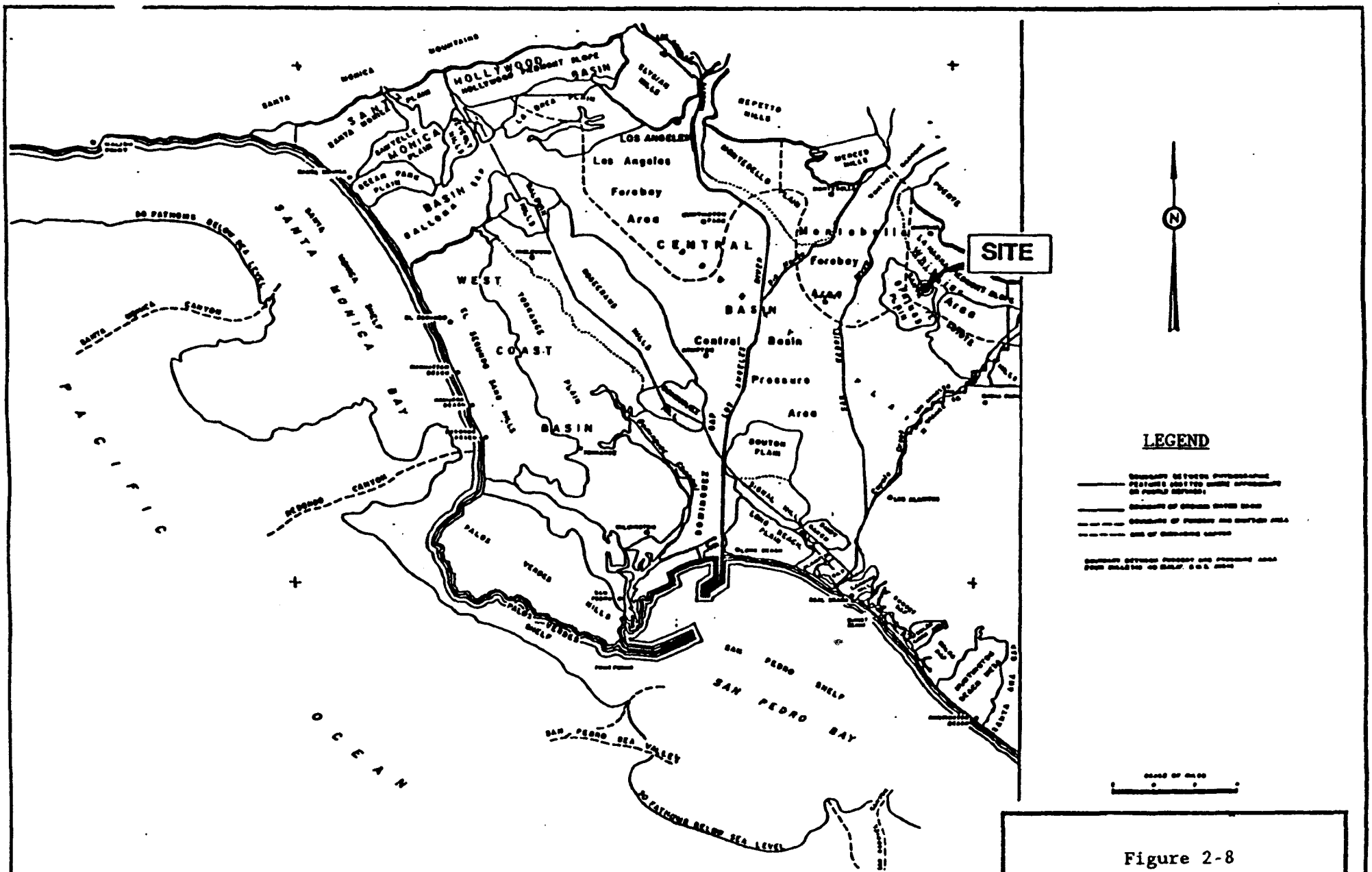


Figure 2-8

GROUNDWATER BASINS OF THE COSTAL PLAIN OF LOS ANGELES COUNTY

SOURCE: Adapted from DWR (1961).

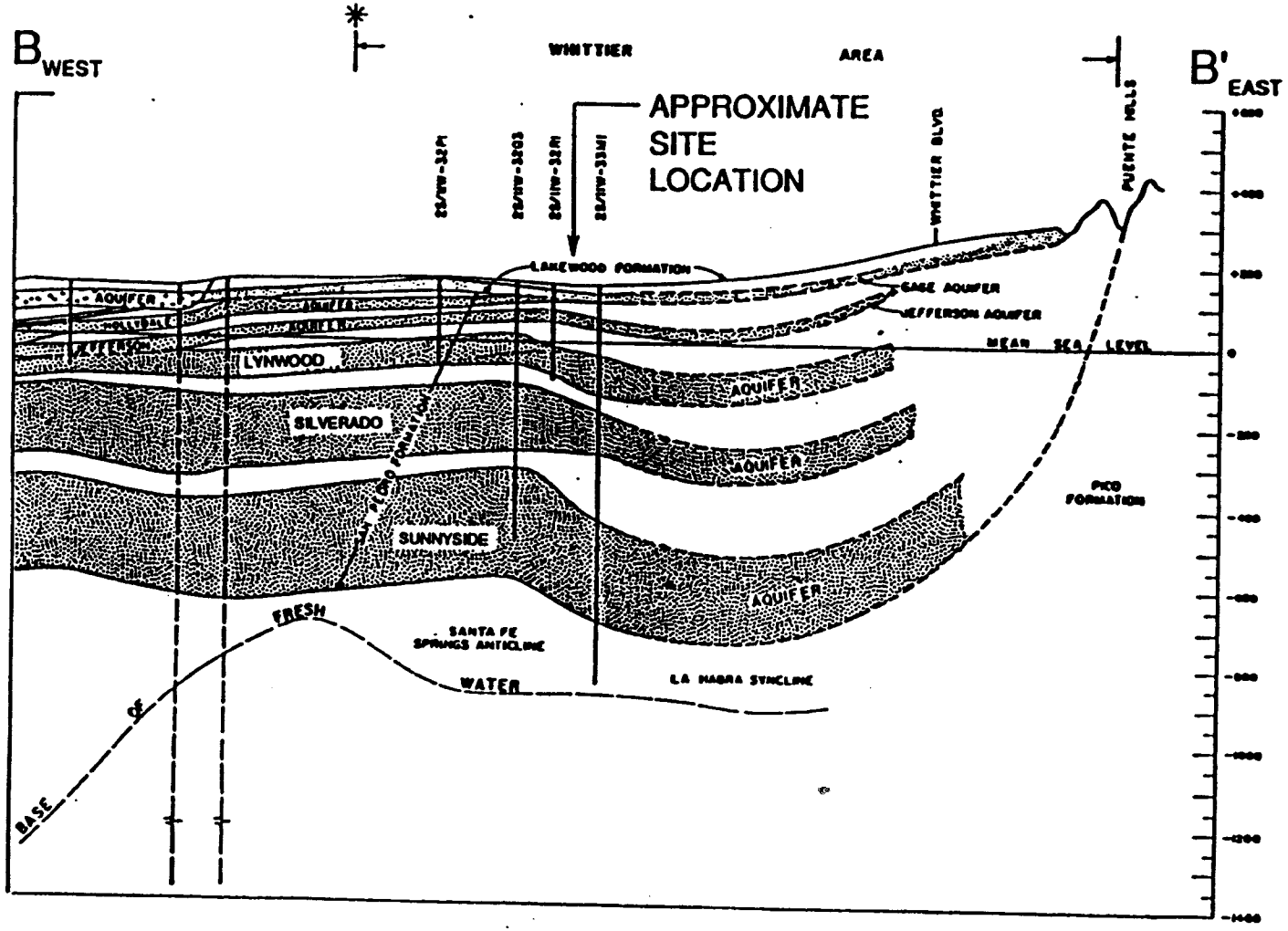


Figure 2-9

WHITTIER AREA CROSS-SECTION VIEW OF WATER BEARING STRATA

SOURCE: Adapted from DWR (1961).

Formation is exposed at the surface, makes it difficult to define the thickness, extent, and composition of the Bellflower aquiclude. Where data are available, the Bellflower aquiclude is clay and sandy clay averaging 20 feet in thickness and extending down to a depth of about 70 feet below the ground surface. According to the Department of Water Resources (1961), the base of the Bellflower aquiclude, as it occurs beneath Santa Fe Springs, is approximately 100 feet above mean sea level (msl).

The degree to which groundwater can be transmitted through the Bellflower aquiclude depends on the thickness and composition of the aquiclude and/or the location and depth of improperly sealed oil and/or water wells. While the aquiclude appears to be continuous over most of the Whittier Area, it may be either absent in some areas or so thin and discontinuous that groundwater can be transmitted though it at an appreciable rate.

In addition to containing the Bellflower aquiclude, the Lakewood Formation also contains the Artesia aquifer, which is mostly sand with some interbedded clay. Near Santa Fe Springs, the maximum thickness of this aquifer is 20 feet. The Gage Aquifer is the major water-bearing member of the Lakewood Formation in the Whittier Area. It has been delineated only in the southern portion of the area and near the Los Angeles-Orange County line, where it consists of about 30 feet of sand with some interbedded clay and has a maximum depth of approximately 150 feet below ground surface (DWR, 1961).

The San Pedro Formation underlies the entire Whittier area, where it attains a maximum thickness of about 850 feet and extends down to a depth of approximately 920 feet. The formation is composed of sand and gravel with interbedded clay, all probably of marine origin. Clay members separate the sands and gravels comprising the aquifers over most of the basin. The San Pedro Formation contains the Hollydale, Jefferson, Lynwood, Silverado, and Sunnyside aquifers. An extensive unconformity brings the aquifers of the San Pedro Formation into contact with those of the Lakewood Formation along the northern boundary of the area and along the edge of the Coyote Hills (EBASCO, 1989c).

The Hollydale aquifer has been identified only in the western part of the Whittier Area. It may be present over the rest of the area, but data are not available to confirm its existence. It ranges in thickness from 10 to 25 feet and consists of sand and gravel with a small amount of interbedded clay. It appears to reach a maximum depth of about 100 feet below ground surface. It is merged with the overlying Gage aquifer in the vicinity of south Whittier. If present beneath the WDI site, the Hollydale aquifer would first be encountered from 85 to 100 feet below ground surface (EBASCO, 1989c).

The Jefferson aquifer ranges in thickness from 20 feet to 40 feet and consists of sand and gravel with a little interbedded clay. It extends over most of the Whittier area and reaches a maximum depth of about 350 feet below ground surface (100 feet below sea level). In the western part of the area, near the boundary with the Montebello Forebay, the Jefferson aquifer merges with the overlying Hollydale aquifer (EBASCO, 1989c).

The Lynwood aquifer is present throughout the Whittier area. It ranges in thickness from 50 to 100 feet and consists of sand and gravel with some interbedded clay. It extends to a maximum depth of about 460 feet below ground surface (EBASCO, 1989c).

The Silverado aquifer has been identified throughout the Whittier area. It consists of 100 to 200 feet of sand and gravel with finger grained phases in some areas. It extends to a depth of about 650 feet below ground surface (EBASCO, 1989c).

The Sunnyside aquifer also has been identified throughout the Whittier area. It consists of 150 to 200 feet of sand and gravel with some interbedded clay. It is the deepest of the aquifers identified, reaching a maximum depth of about 1,000 feet (700 feet below sea level). The gravels exposed in the Coyote Hills and along the north side of the area are believed to be surface outcrops of the Sunnyside aquifer (EBASCO, 1989c).

SITE HYDROGEOLOGY

As discussed in the preceding section, as many as seven aquifers and one aquiclude may be present beneath the WDI site (Figure 2-9). Groundwater at the WDI site is generally located 48 to 65 feet below ground surface. This is approximately 34 to 44 feet below the bottom of the central reservoir and 22 to 47 feet below the bottom of the waste handling areas. The direction of groundwater flow is generally toward the south and west (e.g., from the reservoir and waste handling areas toward the small businesses along Los Nietos Road) (EBASCO, 1989d). Velocity of groundwater flow is unknown and no information is available on other hydrogeologic characteristics of the aquifer(s) beneath the site (EBASCO, 1989c). Monitoring wells installed at the WDI site are believed to have encountered the Bellflower aquiclude at a depth of approximately 50 feet (Dames & Moore, 1985; EBASCO, 1989b). The Gage aquifer is not believed to be present under the WDI site (EBASCO, 1989c).

A total of 27 monitoring wells were installed during the RI of the WDI site. Twenty one (21) of these wells are defined as shallow (approximately 55-70 feet); five (5) are of intermediate depth (approximately 74-130 feet); one well is defined as deep. Additional deep wells were not installed because contamination was not suspected (EBASCO, 1989c).

GROUNDWATER USE

Groundwater use within 3 miles of the WDI site has been classified according to the potential for surrounding populations to ingest contaminants in groundwater from the WDI site as discussed. This approach was outlined in EBASCO's Groundwater Characterization Report (1989c). Of concern are wells utilizing the uppermost aquifer (shallowest groundwater beneath the site), and wells in aquifers which may potentially be hydraulically connected to the uppermost aquifer. Municipal and private groundwater use are discussed below.

Municipal Wells

At present, no nearby wells have been identified which are owned and/or operated by a municipality and draw groundwater from the shallowest water-bearing unit beneath the site.

Although it has not been definitively established that the aquifers in the Lakewood Formation are hydraulically connected to those in the San Pedro Formation, the Department of Water Resources (DWR) concluded that, in the vicinity of the WDI site, the aquifers within each of these formations may be hydraulically connected (DWR, 1961). The possibility that the large number of oil wells in the area and the presence of multiperforated groundwater wells might act as artificial conduits to conduct liquids between aquifers was a major consideration. It is assumed that a well completed in any aquifer possesses a potential for being hydraulically connected to the first aquifer below the WDI site.

The City of Santa Fe Springs provides water for 4200 service connections; these include both residential and industrial hookups. Four out of the five wells which the City of Santa Fe Springs utilizes to obtain 60 percent of its groundwater (40 percent is purchased from the Metropolitan Water District) are located within 3 miles of the WDI site. Both the groundwater which the city pumps and that which it buys are fed directly into the same piping network for distribution (Price, 1985a,b). A small amount of blending is done a city-owned 4 million gallon water storage reservoir.

Private Wells

Two wells of private institutions and individuals which are located within 3 miles upgradient of the WDI site are known to have used the uppermost regional aquifer at some time in the past. Neither of these wells are believed to be currently active. Nine wells which are located within 3 miles of the WDI site, four of which are downgradient of the site, have been

completed in an aquifer which may be hydraulically connected to the uppermost aquifer. These wells are known to have been used at some time in the past by private institutions or individuals. One well is believed to be abandoned, while the status of the other eight is unknown at this time (EBASCO, 1989c).

2.4 CHEMICAL CHARACTERIZATION

To assess the extent of contamination at the site, soil, groundwater, subsurface gas, and air samples were collected at the WDI site and surrounding properties (EBASCO, 1988a,b; 1989a,b,c). Results of sampling efforts and data handling protocols from each of the environmental media are discussed below.

2.4.1 Data Handling Methods

To evaluate chemicals found in environmental media, the following statistical methods were used to assess site conditions. First, the geometric means of chemical concentrations were calculated by EBASCO (1989e) for all environmental media at the site, since the geometric mean, which is the antilogarithm of the arithmetic mean of the logarithms of a set of values, is the most appropriate method to characterize the distribution of chemicals in environmental assessments. This is because environmental chemical data tend to be log-normally distributed (Ott, 1988; Dean, 1981); the geometric mean is the appropriate statistical measure of the central tendency of the lognormal distribution.

Prior to calculating the geometric mean for groundwater and subsurface gas, the geometric means of duplicate samples were calculated and then used in calculating the overall geometric mean for each medium. Frequencies of detection were also calculated subsequent to averaging of duplicates. Duplicate groundwater and subsurface gas samples were considered to be a single sample for the purposes of this report. Replicate soil samples collected at the WDI site varied significantly between the individual sample and the paired replicate. Due to the heterogenous nature of soils, this is

not unusual and therefore, replicate soil samples were considered as discrete sample locations for this assessment.

Two methods were used to calculate geometric means for contaminants at the WDI site. For media suspected to be a contamination source (i.e., a "hot spot"), geometric means were calculated using only the samples where the chemical was positively detected. For those media suspected of being contaminated as a result of contaminant migration, geometric means were calculated using all collected samples, including those samples in which the contaminants were not detected. This assumes that as a result of migration chemicals could be present in concentrations below the analytical detection limit. In this latter case, a value of one-half (½) the analytical detection limit for the sample was used for non-detected samples.

Geometric means were calculated only for chemicals detected in at least one sample. Since soil was assumed to be the source of contamination at the WDI site, only those positively-detected sample locations were used in calculating the geometric mean. For groundwater, contamination is assumed to be dispersing from the soil over the entire area and therefore, geometric means were calculated using all samples. Since both contamination hot spots and areally dispersion are suspected for subsurface gas, the geometric means of contaminated concentrations were calculated using both methods.

2.4.2 Selection of Chemicals of Potential Concern

Based on the results of the RI, a number of chemicals have been identified in the various environmental media at the WDI site. Many of the identified chemicals are associated with petroleum wastes, which are known to have been disposed at the WDI site. These chemicals include benzene, toluene, xylenes, and polycyclic aromatic hydrocarbons (PAHs). As discussed in Section 2.1.1, hazardous wastes were known to be disposed; both to the reservoir and to the ground surface. Therefore, the chemicals found in environmental media

may either have been disposed at the site or be the result of degraded disposed materials.

According to EPA guidance for endangerment assessments conducted for Superfund sites (EPA, 1986a), when a large number of chemicals are detected at a site, a subset of key chemicals of concern is generally selected. The purpose being to focus the risk assessment on those contaminants that are most likely to pose risks to human health and the environment. In accordance with current EPA policies, the following criteria were used to selected the chemicals of concern for the WDI site.

- Comparison with blanks: If chemicals are detected in travel or field blanks, the concentration of compounds in on-site samples and field blanks are compared. On-site concentrations of common laboratory chemicals or field contaminants (e.g., acetone, bis(2-ethylhexyl)phthalate, methylene chloride) that exceed field blank concentrations by ten times and other chemicals that exceed field blank concentrations by five times are considered to be site-related. Sample results not meeting these criteria are generally dropped from further consideration as chemicals of concern.

- Comparison with background concentrations: Inorganic contaminants detected on the site are to be compared to concentrations detected in upgradient samples or in samples from off-site areas that are not contaminated by the site. Typically, chemicals with on-site concentrations that are less than five times the background concentrations are not considered further. This is done by comparing the range reported in the literature to the maximum concentration detected. If only one value for background is available, a factor of five (5x) is used to account for natural variability of inorganic compounds.

- Frequency of detection: When a sufficient number of samples (i.e., at least 20 samples) has been collected to ascertain that the site has been well characterized with respect to any individual chemical, the frequency of detection for any individual chemical can be considered in selecting chemicals of concern. Typically, chemicals detected in less than five percent of the samples are not included as chemicals of concern unless they are known, or potential, carcinogens.

- Consideration of concentration, toxicity, and physicochemical properties: Chemicals with very low toxicity may be removed from further consideration, even though they may meet one of the above criteria for being listed as an indicator chemical of concern.

Conversely, a detected compound which does not meet any of the above criteria, but is known to be highly toxic may be listed as a chemical of concern. Chemicals having high migration potential in the environment may also be selected. EPA Region IX policy requires that all known human carcinogens be carried through the risk assessment process.

The selection processes for the chemicals of concern for each environmental medium are discussed in the following sections.

2.4.2.1 Soils

A total of 255 soil samples were collected during RI activities by EPA's REM III prime contractor (EBASCO, 1989b). Soils at the WDI site were sampled to a maximum depth of 60 feet. Soils to 60 feet in depth at the WDI site were analyzed for volatile organics, semivolatile organics, pesticides, PCBs, and metals. Locations of surface sampling points are shown in Figure 2-10, while subsurface sampling locations are shown in Figure 2-11.

Both organic and inorganic chemicals have been detected in soils at the WDI site. All inorganic elements detected in on-site soils are commonly-occurring soil constituents. For this reason, it is helpful to compare on-site concentrations of these elements to naturally-occurring background concentrations in order to determine whether or not contaminants might be site related. Site-specific background samples were not collected during the RI. Therefore, geometric mean and maximum concentrations of inorganics in surface and subsurface soils were compared to regional background levels, as reported by Shacklette and Boerngen (1984) for the area east of the Palos Verdes Hills and Connor et al. (1975) for the western region in general. These background concentrations are listed in Tables 2-1 and 2-3. Due to natural variability of these inorganic constituents, maximum contaminant concentrations identified as less the five times the maximum background concentration were not considered further as chemicals of concern unless they are known, or probable, human carcinogens. All of those inorganics recognized by EPA as carcinogens were considered as chemicals of concern for the site, regardless of whether they are present in concentrations equal to background.

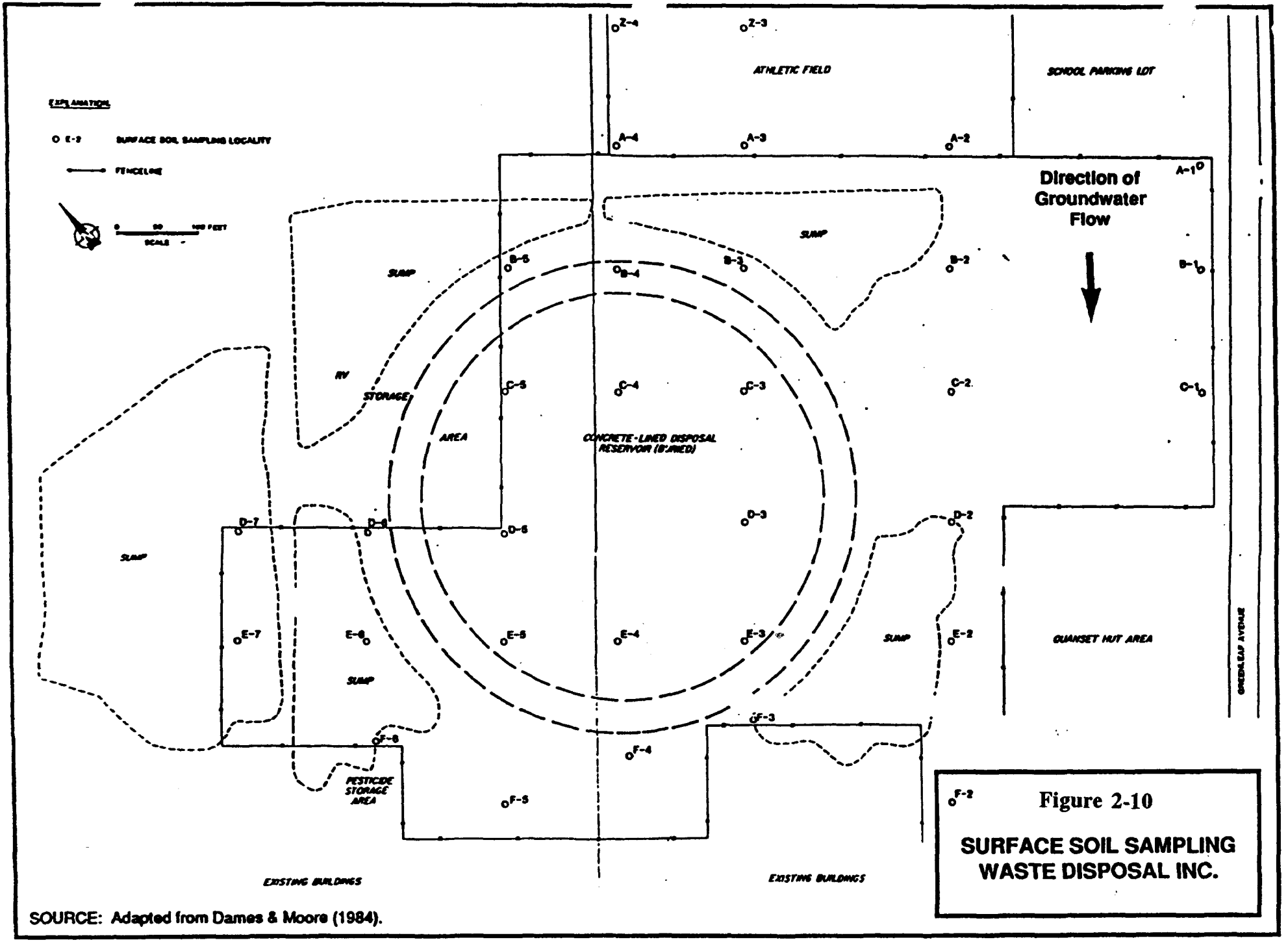


Figure 2-10

SURFACE SOIL SAMPLING
WASTE DISPOSAL INC.

SOURCE: Adapted from Dames & Moore (1984).

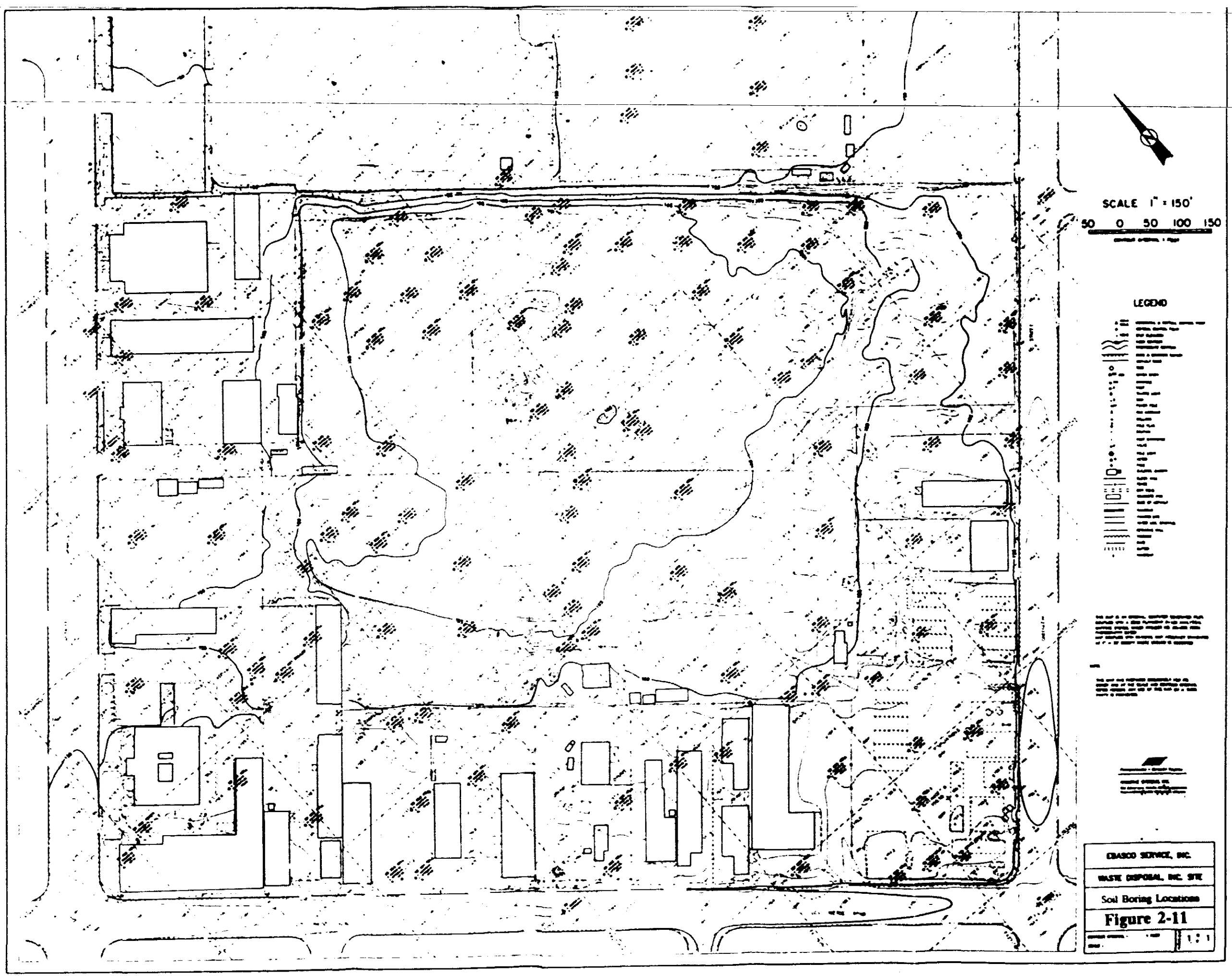

SCALE 1" = 150'
50 0 50 100 150
LEGEND
EXXON SERVICE, INC.
WASTE DISPOSAL, INC. SITE
Soil Boring Locations
Figure 2-11

TABLE 2-1

INORGANICS DETECTED IN SURFACE SOIL SAMPLES
(TO A DEPTH OF 1 FOOT)
COLLECTED AT THE WDI SITE

COMPOUND	NUMBER OF SAMPLES (a)	DETECTION FREQUENCY	CONCENTRATION		
			GEOMETRIC MEAN (b)	MAXIMUM	BACKGROUND (c)
Aluminum	34	100%	12,374	23,700	>100,000
Antimony	34	24%	5.4	7.6	<1
Arsenic	34	100%	8.0	85.1	6.5
Barium	34	100%	204	1690	700
Beryllium	34	85%	0.61	1.2	2-15
Cadmium	34	71%	1.1	3.3	<1-10
Calcium	34	100%	11,123	78,400	18,000-28,000
Chromium	34	100%	23	53	50
Cobalt	34	100%	11	21	15-70
Copper	34	100%	36	511	20
Iron	34	100%	20,409	32,900	30,000
Lead	34	100%	43	731	15
Magnesium	34	100%	6,341	13,600	5,000-7,000
Manganese	34	100%	364	581	500
Mercury	34	62%	0.15	1.2	<0.01-0.02
Molybdenum	34	26%	1.9	3.9	<3
Nickel	34	100%	20	38	20
Potassium	34	100%	3,571	5,190	20,000
Selenium	34	29%	0.50	1.2	0.15-0.20
Silver	34	18%	0.83	4.5	<0.5-5
Sodium	34	88%	423	1,530	15,000-100,000
Thallium	34	15%	7.9	18.7	0.1-0.8
Vanadium	34	100%	40	64.6	150-500
Zinc	34	100%	101	304	74

(a) All soil samples, including replicates, are considered discrete samples.
(b) Geometric mean using all positively detected samples.
(c) Sources: Schacklette and Boerngen, 1984; Conner et al., 1975; Bowen, 1979.

Surface Soils

A total of 34 samples were taken of surface soils at the WDI site. Surface soils are defined as those samples taken to a maximum depth of 12 inches. Tables 2-1 and 2-2 summarize the sample results including the frequency of detection, geometric mean, and maximum concentrations for each detected chemical. Only positively detected samples were used to calculate geometric means for surface soils. A subset of these detected chemicals was selected for use in this using the criteria presented above. Surface soils are presented as a separate unit since these will be evaluated separately from deeper soils.

Inorganic compounds selected as chemicals of concern for WDI surface soils are:

- Antimony,
- Arsenic,
- Cadmium,
- Chromium,
- Copper,
- Lead,
- Mercury,
- Selenium, and
- Thallium.

The maximum concentrations of copper, lead, mercury, selenium, and thallium all exceeded their background concentrations by more than a factor of five. Arsenic and cadmium had geometric mean concentrations within the background range concentrations, but their maximum concentrations exceeded regional background concentrations.

Arsenic and lead were selected as chemicals of concern for surface soils based on their toxicity. The maximum concentrations detected for arsenic and lead exceeded their background concentrations by more than five times. Antimony and thallium were selected based on their high frequencies of

TABLE 2-2
ORGANIC COMPOUNDS DETECTED IN SURFACE SOILS
(TO A DEPTH OF 1 FOOT)
AT THE WASTE DISPOSAL, INC. SITE

Chemical	Count	Frequency of Detection	CONCENTRATION (ug/kg) Geometric Mean	Maximum
CHLORINATED PESTICIDES				
Chlordane (alpha isomer)	4	9%	10	210
DDD	9	18%	66	3300
DDE	12	22%	33	360
DDT	13	25%	37	280
Dieldrin	4	9.3%	38	280
Endosulfan II	1	2.3%	120	120
Heptachlor epoxide	2	7.0%	9.6	46
MONOCYCLIC AROMATIC HYDROCARBONS				
Benzene	1	2.2%	260	260
Ethylbenzene	6	14%	47	1,500
Toluene	43	88%	52	9,000
1,2,4-Trichlorobenzene	1	2.0%	35	35
Xylenes	5	11%	190	2,500
ALIPHATIC HYDROCARBONS				
Chloroform	1	2.6%	5	5
1,2-Dichloroethylene	1	2.8%	4	4
Methylene chloride	9	39%	23	1,400
Vinyl Acetate	1	2.6%	9	9
KETONES				
Acetone	4	15%	35	120
2-Butanone	16	32%	8.2	46
Isophorone	1	2.0%	150	150
4-Methyl-2-Pentanone	1	2.3%	7	7
PHTHALATES				
Bis(2-ethylhexyl)phthalate	13	24%	280	830,000
Butylbenzylphthalate	6	12%	450	17,000
Di-n-butylphthalate	8	15%	170	1,800
Dimethylphthalate	1	2.0%	1,000	1,000
Di-n-octylphthalate	4	7.1%	950	88,000
PHENOLS				
4-Chloro-3-methylphenol	2	4.0%	90	110
2-Methylphenol	1	2.0%	79	79
4-Methylphenol	1	2.0%	110	110
4-Nitrophenol	2	4.0%	1,000	1,700
Phenol	1	2.0%	2,000	2,000
Pentachlorophenol	3	6.0%	250	290

TABLE 2-2 -CONTINUED-

ORGANIC COMPOUNDS DETECTED IN SURFACE SOILS
(TO A DEPTH OF 1 FOOT)
AT THE WASTE DISPOSAL, INC. SITE

Chemical	Count	Frequency of Detection	CONCENTRATION (ug/kg) Geometric Mean	Maximum
POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)				
Carcinogenic				
Benzo(a)anthracene	5	8.1%	140	380
Benzo(a)pyrene	7	11%	220	750
Benzo(b)fluoranthene	5	8.1%	200	350
Benzo(k)fluoranthene	3	6.0%	130	340
Chrysene	6	10%	200	600
Noncarcinogenic				
Acenaphthene	1	2.0%	740	740
Anthracene	2	3.8%	180	620
Benzo(g,h,i)perylene	1	1.9%	530	530
Dibenzofuran	1	2.0%	400	400
Fluoranthene	9	15%	150	520
Fluorene	2	3.7%	440	1,400
2-Methylnaphthalene	8	14%	600	6,800
Naphthalene	4	7.1%	960	3,100
Phenanthrene	5	8.9%	270	2,700
Pyrene	12	19%	150	1,100
POLYCHLORINATED BIPHENYLS (PCBs)				
Aroclor-1248	1	2.3%	1,500	1,700
Aroclor-1260	2	4.7%	570	3,200
ORGANIC ACIDS				
Benzoic Acid	3	6.0%	150	230

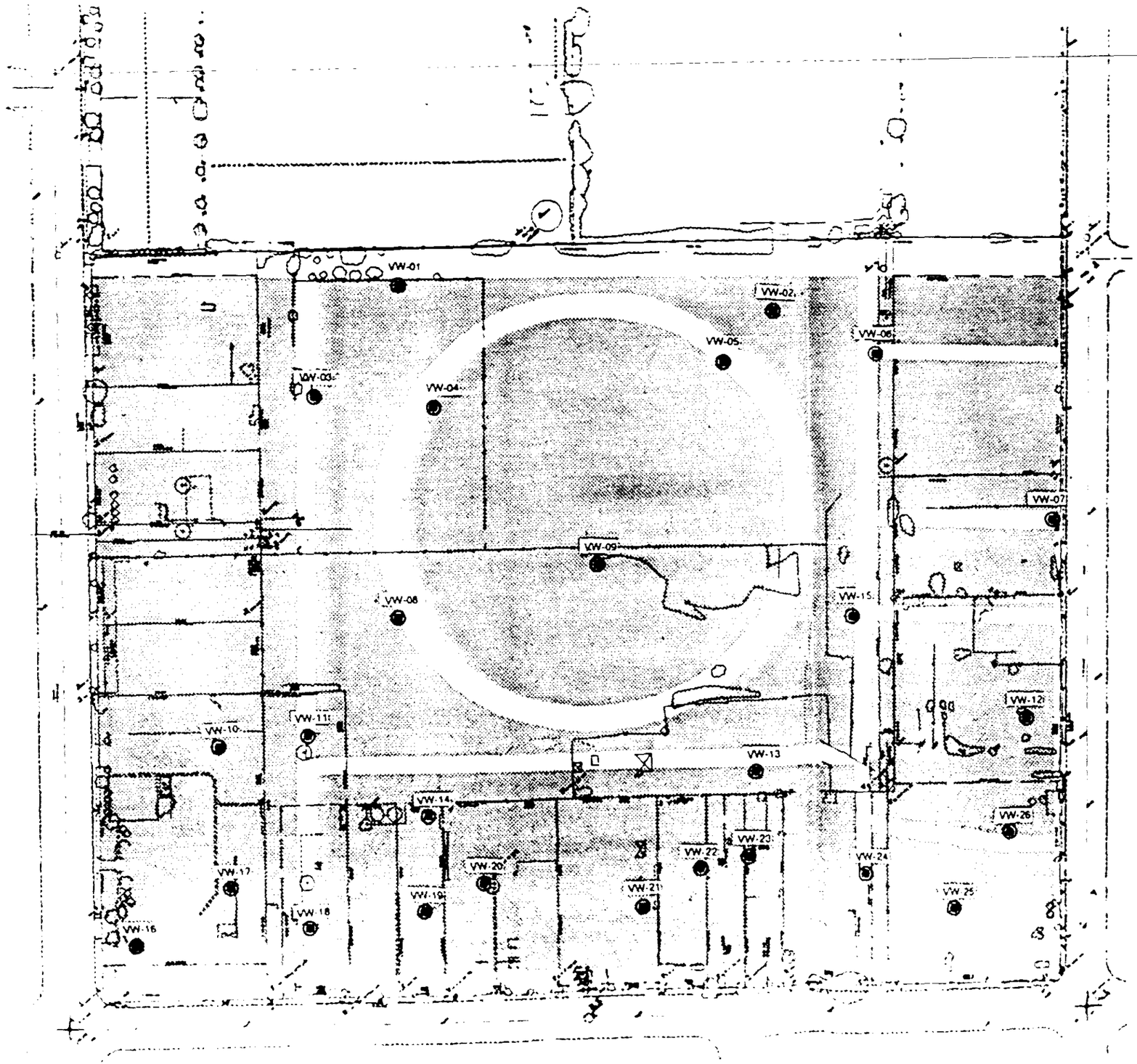
detection and comparison to background concentrations. Calcium and magnesium are necessary human nutrients (NAS, 1976a) and, so, were eliminated from consideration. Concentrations of aluminum, barium, beryllium, cobalt, iron, magnesium, manganese, molybdenum, nickel, potassium, sodium, vanadium, and zinc were detected frequently (i.e., greater than 70% of all samples) but did not exceed their background concentrations by greater than five times, thus none of these inorganic elements were selected as chemicals of potential concern. Silver was detected in 20 percent of all surface soil samples and was detected in concentrations which did not exceed regional background concentrations by more than 5 times. Therefore, silver will not be considered a chemical of potential concern in this assessment.

Organic compounds detected in soils were grouped by chemical class (see Table 2-2). These chemicals can be classified into eight groups based on their structure. These eight groups are: aromatic acids (e.g., benzoic acid), chlorinated aliphatic hydrocarbons (e.g., methylene chloride), chlorinated pesticides (e.g., DDT), ketones (e.g., 2-butanone), monocyclic aromatic chemicals (e.g., toluene), phenols (e.g., pentachlorophenol), polycyclic aromatic hydrocarbons (e.g., benzo(a)pyrene, naphthalene), and polychlorinated biphenyls (e.g., Aroclor 1260).

Chlorinated pesticides selected as chemicals of potential concern are:

- Chlordane (α and -isomers),
- DDT, DDD, DDE,
- Dieldrin, and
- Heptachlor epoxide.

Chlordane, DDT, DDD, DDE, dieldrin, and heptachlor epoxide, were all associated with Toxo Spray Dust activities, have known carcinogenic potential, and were detected in greater than 5 percent of the samples collected. In future discussions, DDT will refer to the sum of the DDT, DDD, and DDE concentrations detected.



LEGEND

- HORIZONTAL & VERTICAL CONTROL POINT
- VERTICAL CONTROL POINT
- SPOT ELEVATION
- INDEX CONTOUR
- INTERMEDIATE CONTOUR
- CURB & CONCRETE GUTTER
- ASPHALT ROAD
- TREE
- CATCH BASIN
- MANHOLE
- POST
- TRAFFIC LIGHT
- SIGN
- POWER POLE
- FIRE HYDRANT
- BOLLARD
- FIRE FLAG
- BOLLARD
- POST BARRICADE
- VALVE
- POLE LIGHT
- METER
- POLE
- BUILDING, CANOPY
- BLOCK WALL
- FENCE
- DIRT ROAD
- CONCRETE PAD
- EDGE OF ASPHALT
- RAILROAD
- PAINTED LINE
- WATER LINE, DRAINAGE
- RETAINING WALL
- TREELINE
- GATE
- GUTTER
- HANDICAP
- GAS MONITORING WELL

SOIL BORING NUMBERS
FOR VADOSE WELLS

VW-01=SB-009	VW-14=SB-084
VW-02=SB-019	VW-15=SB-060
VW-03=SB-023	VW-16=SB-080
VW-04=SB-035	VW-17=SB-081
VW-05=SB-027	VW-18=SB-082
VW-06=SB-028	VW-19=SB-083
VW-07=SB-052	VW-20=SB-085
VW-08=SB-056	VW-21=SB-086
VW-09=SB-048	VW-22=SB-087
VW-10=SB-064	VW-23=SB-088
VW-11=SB-065	VW-24=SB-089
VW-12=SB-071	VW-25=SB-090
VW-13=SB-077	VW-26=SB-079

SCALE
APPROX MATE

EBASCO SERVICES, INC.

WASTE DISPOSAL, INC. SITE

Figure 2-13

LOCATION OF
SUBSURFACE GAS WELLS

TABLE 2-6
SUBSURFACE GAS SAMPLE RESULTS
COLLECTED AT THE WDI SITE

Chemical	Detection Frequency	Total Samples	CONCENTRATION (in ug/m3) Geometric Mean (a)	Geometric Mean (b)	Maximum	Detection Limit	Blank Geometric Mean (c)
Benzene	38%	26	99	524	28,000	64	67
Carbon Tetrachloride	3.8%	26	1.7	9.4	9.4	3.1	ND (d)
Chloroform	15%	26	3.8	45	120	4.9	ND
1,2-Dibromoethane	81%	26	160	209	590	110	210
1,2-Dichloroethane	3.8%	26	42	117	120	81	ND
Tetrachloroethylene	100%	26	88	88	520	6.8	30
1,1,1-Trichloroethane	50%	26	44	130	6,300	27	39
Trichloroethylene	92%	26	150	215	16,000	5.4	9.1
Vinyl Chloride	12%	26	38	945	20,000	51	ND

(a) - Geometric mean using all positively detected samples and one half the detection limit for non-detected samples.
(b) - Geometric mean using positive detects only.
(c) - Two blank samples were collected.
(d) - ND = Not detected in either blank sample.

Chemicals of concern selected for subsurface gas are:

- Benzene,
- Carbon tetrachloride,
- Chloroform,
- 1,2-Dibromoethane,
- 1,2-Dichloroethane,
- Tetrachloroethylene,
- 1,1,1-Trichloroethane,
- Trichloroethylene, and
- Vinyl chloride.

Tetrachloroethylene (PCE) was detected in all gas samples including blank samples. The geometric mean and maximum concentrations for field samples are 88 and 520 $\mu g/m^3$, respectively; the geometric mean concentration of tetrachloroethene in the blank samples is 30 $\mu g/m^3$. Other frequently detected chemicals include 1,1,1-trichloroethane (50%), 1,2-dibromoethane (81%), benzene (38%), and trichloroethene (92%). Vinyl chloride was detected in three of 26 samples at a geometric mean concentration of 38 $\mu g/m^3$ and a maximum concentration of 20,000 $\mu g/m^3$. Chloroform was found in four of 26 samples. Carbon tetrachloride and 1,2-dichloroethane were each detected in 1 of the 26 samples.

Benzene and 1,2-dibromoethane are associated with petroleum wastes. 1,2-Dibromoethane (EDB) is commonly added to leaded gasoline as an antifouling agent. 1,1,1-Trichloroethane, carbon tetrachloride, tetrachloroethylene, and trichloroethylene are all used as degreasers which may have been disposed at the WDI facility. Vinyl chloride is a product of biological degradation of these chlorinated degreasers.

Benzene, 1,2-dibromoethane, 1,2-dichloroethane, carbon tetrachloride, chloroform, tetrachloroethylene, trichloroethylene, and vinyl chloride all have known carcinogenic potential.

2.4.2.4 Air

Airborne particulate samples were collected by EBASCO during the RI sampling effort. However due to problems in the laboratory analysis, these data were of insufficient quality for data validation. Therefore, these data are not presented or used in this risk assessment.

2.4.3 Summary

As discussed above, chemicals have been detected in all environmental media sampled at the site and a subset of these chemicals have been selected as chemicals of potential concern for these media. Inorganic compounds were selected as chemicals of concern for surface and subsurface soils. Arsenic, lead, manganese, and mercury were also selected as chemicals of concern for groundwater. Inorganics were not selected as chemicals of concern for subsurface gas. Many chlorinated pesticides were selected as chemicals of concern for surface and subsurface soils. Aldrin, BHC, chlordane, and heptachlor were included only in subsurface soils. Chlorinated pesticides were not considered for subsurface gas.

The monocyclic aromatic hydrocarbons were selected as chemicals of concern for all media. Benzene, ethylbenzene, toluene, and xylenes were found in surface and subsurface soils. Toluene was the most detected organic contaminant in groundwater, and benzene was also selected as a chemical of concern for subsurface gas.

Benzoic acid was only detected in surface and subsurface soils and was selected as a chemical of concern for surface soils. 2-Butanone was detected in surface and subsurface soils and was selected as a chemical of concern for both.

Polycyclic aromatic hydrocarbons (PAHs) and polychlorinated biphenyls (PCBs) were detected in surface and subsurface soils and were selected as

chemicals of concern. Neither PAHs nor PCBs were detected in groundwater. PAHs and PCBs were not analyzed in subsurface gas.

Halogenated aliphatic hydrocarbons were detected in subsurface soils, groundwater, and subsurface gas. Chloroform, tetrachloroethylene, and trichlorethylene were selected as chemicals of concern for all media. 1,2-Dibromoethane and 1,2-dichloroethane were detected only in subsurface gas.

A summary of selected chemicals of concern and environmental media is presented in Table 2-7.

TABLE 2-7
CHEMICALS OF POTENTIAL CONCERN FOR ALL MEDIA
WDI SITE

Compound	Surface Soils	Soils 0 to 20 feet	Groundwater	Subsurface Gas
INORGANICS				
Antimony	X	X		
Arsenic	X	X	X	
Cadmium	X	X		
Chromium	X	X		
Copper	X	X		
Lead	X	X	X	
Manganese		X	X	
Mercury	X	X	X	
Selenium	X	X		
Thallium	X	X		
Zinc		X		
CHLORINATED PESTICIDES				
Aldrin		X		
gamma-BHC (Lindane)		X		
Chlordane	X	X		
DDT, DDD, DDE	X	X		
Dieldrin	X	X		
Heptachlor		X		
Heptachlor Epoxide	X	X		
PHENOLS				
Pentachlorophenol	X	X		
MONOCYCLIC AROMATIC HYDROCARBONS				
Benzene	X	X		X
1,4-Dichlorobenzene		X		
Ethylbenzene	X	X		
Toluene	X	X	X	
Xylenes	X	X		
ORGANIC ACIDS				
Benzoic Acid	X	X		
POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)				
--carcinogenic	X	X		
--noncarcinogenic	X	X		
POLYCHLORINATED BIPHENYLS (PCBs)	X	X		

TABLE 2-7 (continued)
CHEMICALS OF POTENTIAL CONCERN FOR ALL MEDIA
WDI SITE

Compound	Surface Soils	Soils 0 to 20 feet	Groundwater	Subsurface Gas
KETONES				
2-butanone	X	X		
HALOGENATED ALIPHATIC HYDROCARBONS				
Carbon Tetrachloride		X	X	X
Chloroform		X	X	X
1,2-Dibromoethane				X
1,2-Dichloroethane				X
Methylene Chloride	X	X		
Tetrachloroethylene		X	X	X
1,1,1-Trichloroethane		X		X
Trichloroethylene		X	X	X
Vinyl chloride		X		X

3.0 TOXICITY CHARACTERIZATION

In this section, brief descriptions of the toxic properties of the chemicals of potential concern at the WDI site are presented together with available standards, criteria, and toxicity values which have been developed for evaluation of exposure to these chemicals. The health-based criteria that will be used in the quantitative risk estimation are summarized in Tables 3-1 and 3-2.

3.1 HEALTH EFFECTS CLASSIFICATION AND CRITERIA DEVELOPMENT

For risk assessment purposes, chemicals are separated into two categories of toxicity, depending on whether they exhibit noncarcinogenic or carcinogenic effects. This distinction relates to the currently-held scientific opinion that the mechanisms of action for these categories differ. For carcinogens, it is assumed that there is no level of exposure which does not have a finite possibility of causing the disease (i.e., there is no threshold dose for carcinogenic effects). For chemicals exhibiting noncarcinogenic effects, it is believed that organisms have protective mechanisms that must be overcome before the toxic endpoint is produced (i.e., there is a threshold dose for these effects). For example, if a large number of cells performs the same or similar functions, it would be necessary for significant damage or depletion of these cells to occur before a toxic effect could be seen. This threshold view for noncarcinogenic effects holds that a range of exposures up to a defined threshold can be tolerated by the organism without appreciable risk of causing the disease (EPA, 1986d).

3.1.1 HEALTH EFFECTS CRITERIA FOR POTENTIAL NONCARCINOGENS

Reference doses (RfDs) developed by the EPA RfD Work Group or obtained from Health Effects Assessments (HEAs) are generally used as health criteria for chemicals exhibiting noncarcinogenic effects. The RfD, expressed in units of mg/kg/day, is an estimate of the maximum human chronic daily exposure level

TABLE 3-1
CRITICAL ORAL TOXICITY VALUES
CHEMICALS OF POTENTIAL CONCERN
WASTE DISPOSAL INC.

Chemical	RfD (mg/kg/day) [Uncertainty Factor][a]	Cancer Potency Factor $(mg/kg/day)^{-1}$	EPA Weight of Evidence[b]	Source[c]
ORGANICS				
Aldrin	3×10^{-5} [1000]	17	B2	IRIS/HEA
Benzene	--	2.9×10^{-2}	A	IRIS
Benzene Hexachloride (BHC)				
technical-grade	--	1.8	B2	IRIS
alpha-isomer	--	6.3	B2	IRIS
beta-isomer	--	1.8	C	IRIS
gamma-isomer	3×10^{-4} [1000]	1.3	B2	IRIS
Benzoic acid	4 [1]	--	--	IRIS
2-Butanone	5×10^{-2} [1000]	--	--	IRIS
Carbon tetrachloride	7×10^{-4} [1000]	1.3×10^{-1}	B2	IRIS
Chlordane	6×10^{-5} [1000]	1.3	B2	IRIS
Chloroform	1×10^{-2} [1000]	6.1×10^{-3}	B2	IRIS
DDT	5×10^{-4} [100]	3.4×10^{-1}	B2	IRIS
1,2-Dibromoethane	--	85	B2	IRIS
1,4-Dichlorobenzene	1×10^{-1} [1000]	2.4×10^{-2}	B2	HA
1,2-Dichloroethane	--	9.1×10^{-2}	B2	IRIS
Dieldrin	5×10^{-5} [100]	16	B2	IRIS
Ethylbenzene	1×10^{-1} [1000]	--	--	IRIS
Heptachlor	5×10^{-4} [300]	4.5	B2	IRIS
Heptachlor Epoxide	1.3×10^{-5} [1000]	9.1	B2	IRIS
Methylene Chloride	6×10^{-2} [100]	7.5×10^{-3}	B2	HEA
Pentachlorophenol	3×10^{-2} [100]	1.6×10^{-2}	B2	HEA/DHS
Polychlorinated Biphenyls	--	7.7	B2	IRIS
Polycyclic Aromatic Hydrocarbons				
noncarcinogenic	4.1×10^{-1} [100]	--	--	HEA
carcinogenic	--	11.5	B2	HEA
Tetrachloroethylene	1×10^{-2} [1000]	5.1×10^{-2}	B2	IRIS/HEA
Toluene	3×10^{-1} [100]	--	--	IRIS
1,1,1-Trichloroethane	9×10^{-2} [1000]	--	--	IRIS
Trichloroethylene	7.35×10^{-3}	1.1×10^{-2}	B2	HEA

TABLE 3-1 (cont.)
CRITICAL ORAL TOXICITY VALUES
CHEMICALS OF POTENTIAL CONCERN
WASTE DISPOSAL INC.

Chemical	RfD (mg/kg/day)	[Uncertainty Factor][a]	Cancer Potency Factor $(mg/kg/day)^{-1}$	EPA Weight of Evidence[b]	Source[c]
Vinyl Chloride	--		2.3	A	IRIS
Xylenes	2	[100]	--	--	HEA
INORGANICS					
Antimony	4×10^{-4}	[1000]	--	--	IRIS
Arsenic	1×10^{-3}		2.0	A	EPA, 1988d
Cadmium					
Drinking water exposure	5×10^{-4}		--	--	HEA
Other oral routes	1×10^{-3}		--	--	HEA
Chromium (III)	1.0	[1000]	--	--	IRIS
Chromium (VI)	5×10^{-3}	[500]	--	--	IRIS
Copper	3.7×10^{-2}	[2]	--	--	EPA, 1987i*
Lead	6×10^{-4}		--	B2	--
Manganese	2×10^{-1}	[100]	--	--	HEA
Mercury, inorganic	3×10^{-4}	[1000]	--	--	HEA
Mercury, organic	3×10^{-4}	[10]	--	--	HEA
Selenium	3×10^{-3}	[15]	--	--	HEA
Thallium	7×10^{-5}	[3000]	--	--	HEA
Zinc	2.1×10^{-1}	[10]	--	--	IRIS

[a] Uncertainty factors used to develop reference doses consist of multiples of 10, each factor representing a specific area of uncertainty inherent in the data available. The standard uncertainty factors include:

- a ten-fold factor to account for the variation in sensitivity among the members of the human population;
- a ten-fold factor to account for the uncertainty in extrapolating animal data to the case of humans;

TABLE 3-1 (cont.)
CRITICAL ORAL TOXICITY VALUES
CHEMICALS OF POTENTIAL CONCERN
WASTE DISPOSAL INC.

- a ten-fold factor to account for the uncertainty in extrapolating from less than chronic NOELs to chronic NOAELs; and
- a ten-fold factor to account for uncertainty in extrapolating from LOAELS to NOAELs.

[b] Weight of evidence classification schemes for carcinogens: A – Human Carcinogen, sufficient evidence from human epidemiological studies; B1 – Probable Human Carcinogen, limited evidence from epidemiological studies and adequate evidence from animal studies; B2 – Probable Human Carcinogen, inadequate evidence from epidemiological studies and adequate evidence form animal studies; C – Possible Human Carcinogen, limited evidence in animals in the absence of human data; D – Not Classified as to human carcinogenicity; and E – Evidence of Noncarcinogenicity.

[c] Sources: IRIS - Integrated Risk Information System; HEA - Health Effects Assessment; DHS - California Department of Health Services; EPA, 1987i, 1988d.

* Current Federal Drinking Water Standard (MCL and MCLG) of 1.3 mg/L; assuming a 70 kg person ingests 2 liters of water per day; 1.3 mg/l = 0.037 mg/kg/day.

TABLE 3-2
CRITICAL INHALATION TOXICITY VALUES
FOR CHEMICALS OF POTENTIAL CONCERN
WASTE DISPOSAL INC.

Chemical	RfD (mg/kg/day) [Uncertainty Factor][a]	Cancer Potency Factor $(mg/kg/day)^{-1}$	EPA Weight of Evidence[b]	Source[c]
ORGANICS				
Aldrin	--	17	B2	IRIS
Benzene	--	2.9×10^{-2}	A	IRIS
Benzene Hexachloride (BHC)				
technical-grade	--	1.8	B2	IRIS
alpha-isomer	--	6.3	B2	IRIS
beta-isomer	--	1.8	C	IRIS
gamma-isomer	--	--		IRIS
Benzoic acid	--	--	--	IRIS
2-Butanone	9×10^{-2} [1000]	--	--	HEA
Carbon tetrachloride	--	1.3×10^{-1}	B2	IRIS
Chlordane	--	1.3	B2	IRIS
Chloroform	--	8.1×10^{-2}	B2	IRIS
DDT	--	0.34	B2	IRIS
1,2-Dibromoethane	--	7.6×10^{-1}	B2	IRIS
1,4-Dichlorobenzene	2×10^{-1} [100][d]	--	B2	HEA
1,2-Dichloroethane	--	9.1×10^{-2}	B2	IRIS
Dieldrin	--	16	B2	IRIS
Ethylbenzene	1×10^{-1} [e]	--	--	--
Heptachlor	--	4.5	B2	IRIS
Heptachlor Epoxide	--	9.1	B2	IRIS
Methylene Chloride	--	1.4×10^{-2}	B2	HEA
Pentachlorophenol	3×10^{-2} [100]	1.6×10^{-2}	B2	HEA/DHS
Polychlorinated biphenyls	--	--	--	--
Polycyclic Aromatic Hydrocarbons				
noncarcinogenic	4.1×10^{-1} [d]	--	--	HEA
carcinogenic	--	6.1	B2	HEA
Tetrachloroethylene	--	3.3×10^{-3}	B2	HEA
Toluene	1 [100]	--	--	HEA
1,1,1-Trichloroethane	3×10^{-1} [1000]	--	--	IRIS
Trichloroethylene	--	1.3×10^{-2}	B2	HEA

TABLE 3-2 (cont.)
CRITICAL INHALATION TOXICITY VALUES
FOR CHEMICALS OF POTENTIAL CONCERN
WASTE DISPOSAL INC.

Chemical	RfD (mg/kg/day) [Uncertainty Factor][a]	Cancer Potency Factor $(mg/kg/day)^{-1}$	EPA Weight of Evidence[b]	Source[c]
Vinyl Chloride	--	2.95×10^{-1}	A	IRIS
Xylenes	3×10^{-1} [1000]	--	--	HEA
INORGANICS				
Antimony	4×10^{-4} [e]	--	--	--
Arsenic	--	50	A	IRIS
Cadmium	--	6.1	B1	IRIS
Chromium (III)	--	--	--	--
Chromium (VI)	--	41	A	IRIS
Copper	1×10^{-2} [e] [10]	--	--	EPA, 1987i
Lead	6×10^{-4} [e]	--	--	B2
Manganese	3×10^{-4} [100]	--	--	HEA
Mercury, inorganic	5.1×10^{-5} [e] [100]	--	--	--
Mercury, organic	1×10^{-4} [10]	--	--	--
Selenium	1×10^{-3} [10]	--	--	HEA
Thallium	--	--	--	--

[a] Uncertainty factors used to develop reference doses consist of multiples of 10, each factor representing a specific area of uncertainty inherent in the data available. The standard uncertainty factors include:
- a ten-fold factor to account for the variation in sensitivity among the members of the human population;
- a ten-fold factor to account for the uncertainty in extrapolating animal data to the case of humans;
- a ten-fold factor to account for the uncertainty in extrapolating from less than chronic NOELs to chronic NOAELs; and
- a ten-fold factor to account for uncertainty in extrapolating from LOAELS to NOAELs.

TABLE 3-2 (cont.)
CRITICAL INHALATION TOXICITY VALUES
FOR CHEMICALS OF POTENTIAL CONCERN
WASTE DISPOSAL INC.

[b] Weight of evidence classification schemes for carcinogens: A – Human Carcinogen, sufficient evidence from human epidemiological studies; B1 – Probable Human Carcinogen, limited evidence from epidemiological studies and adequate evidence from animal studies; B2 – Probable Human Carcinogen, inadequate evidence from epidemiological studies and adequate evidence from animal studies; C – Possible Human Carcinogen, limited evidence in animals in the absence of human data; D – Not Classified as to human carcinogenicity; and E – Evidence of Noncarcinogenicity.

[c] Sources: IRIS - Integrated Risk Information System; HEA - Health Effects Assessment; DHS - California Department of Health Services.

[d] Inhalation RfD for 1,4-dichlorobenzene calculated from inhalation RfD of 7.1×10^{-1} mg/m^3 using an inhalation rate of 20 m^3/day and a body weight of 70kg.

[e] Values for oral RfDs have been used when inhalation RfDs were not available, to estimate risks due to inhalation of these chemicals in this assessment.

that is likely to be without an appreciable risk of deleterious effects during a lifetime. RfDs are usually derived either from human studies involving workplace exposures or from animal studies, and are adjusted using uncertainty factors, as described in Barnes et al. (1987). The RfD provides a benchmark against which human intakes of chemicals estimated for exposures to contaminated environmental media may be compared. EPA has derived RfDs for several of the chemicals of potential concern at WDI.

3.1.2 HEALTH EFFECTS CRITERIA FOR POTENTIAL CARCINOGENS

Cancer potency factors are generally used as health criteria for potentially carcinogenic chemicals. They are derived from the results of chronic animal bioassays or human epidemiological studies and are expressed in units of $(mg/kg/day)^{-1}$. Animal bioassays are usually conducted at dose levels that are much higher than those likely to be produced by human exposure to environmental media, in order to detect possible adverse effects in the small test populations used in these studies. Since humans are generally exposed at lower doses, the data are adjusted using mathematical models. A linearized multistage model is typically fitted to data from animal studies to obtain a dose-response curve (Crump et al., 1976). The 95th percentile, upper confidence limit slope of the linear term in the dose-response curve (q^*) is subjected to various adjustments and an interspecies scaling factor is usually applied to derive a cancer potency factor for humans. Dose-response data derived from human epidemiological studies are fitted to dose-time-response curves on an ad hoc basis. In both types of analysis, conservative assumptions are applied and the models provide rough, but plausible, estimates of the upper limits on lifetime risk as a function of dose. The actual risks associated with exposure to a potential carcinogen quantitatively evaluated on the basis of a cancer potency factor are not likely to exceed the risks estimated given reasonable exposure estimates, but may be much lower.

EPA also assigns weight-of-evidence classifications to potential carcinogens. Under this system, chemicals are classified as either Group A, Group B1, Group B2, Group C, Group D, or Group E. Group A chemicals (known

human carcinogens) are agents for which there is sufficient evidence to support the causal association between exposure to the agents in humans and cancer. Groups B1 and B2 chemicals (probable human carcinogens) are agents for which there is limited (B1), or inadequate (B2) evidence of carcinogenicity from human studies, but for which there is sufficient evidence of carcinogenicity from animal studies. Group C chemicals (possible human carcinogens) are agents for which there is limited evidence of carcinogenicity in animals, and Group D chemicals (not classified as to human carcinogenicity) are agents with inadequate human and animal evidence of carcinogenicity or for which no data are available. Group E chemicals (evidence of non-carcinogenicity in humans) are agents for which there is no evidence of carcinogenicity in adequate human or animal studies.

3.2 TOXICITY OF THE CHEMICALS OF CONCERN

Brief descriptions of the toxicity of the chemicals of concern at the WDI site are presented below.

ORGANIC CHEMICALS

3.2.1 ALDRIN

Aldrin is absorbed by humans following oral, inhalation, or dermal exposure (Deichmann, 1981; Feldman and Maibach, 1974; NIOSH, 1978a). It is metabolically converted to and stored as dieldrin in fatty tissues (Bann et al., 1956); therefore, the toxicities of aldrin have been considered to be the same as dieldrin (ACGIH, 1986). Both compounds affect primarily the central nervous system. Reported effects in humans following acute exposure include malaise, incoordination, headache, dizziness, gastrointestinal disturbances and major motor convulsions (NRC, 1982). No adverse effects were noted in humans chronically exposed to doses up to 3 mg/kg/day (Houk and Robinson, 1967; Hunter et al., 1969). In laboratory animals, acute exposure produces irritability, tremors, and convulsions (Heath and Vandekar, 1964); chronic exposure results in hepatic and renal toxicity (Treon and Cleveland, 1955;

Walker et al., 1969; Fitzhugh et al., 1964). Aldrin has been shown to be fetotoxic and/or teratogenic in hamsters and mice (Ottolenghi et al., 1974). It had marked effects on fertility, gestation, viability, and lactation in a six-generation mouse study (Deichmann, 1972).

Data relating to the carcinogenicity of aldrin in humans are limited. Aldrin has been shown to produce liver tumors in mice following chronic oral exposure (NCI, 1978a). In contrast, chronic feeding studies with aldrin in rats have generally yielded negative results with respect to carcinogenicity (NCI, 1978a; Fitzhugh et al., 1964). EPA (1989a) classified aldrin in Group B2 – Probable Human Carcinogen, with oral and inhalation cancer potency factors of 17 $(mg/kg/day)^{-1}$. These values are based on feeding studies in mice in which increased incidences of liver carcinoma were observed (Davis, 1965 reevaluated by Reuber as cited by Epstein, 1976; NCI, 1978a).

EPA (1989a) reported an oral RfD for aldrin of 3×10^{-5} mg/kg/day based on a chronic feeding study by Fitzhugh et al. (1964) in which liver lesions were observed in rats administered 0.5 ppm (0.025 mg/kg/day) aldrin in the diet; an uncertainty factor of 1000 was used to develop the RfD.

3.2.2 BENZENE

Benzene is readily absorbed following oral and inhalation exposure (EPA, 1985a). The toxic effects of benzene in humans and other animals following exposure by inhalation include central nervous system effects, hematological effects, and immune system depression (ATSDR, 1987a). In humans, acute exposure to high concentrations of benzene vapors has been associated with dizziness, nausea, vomiting, headache, drowsiness, narcosis, coma, and death (NAS, 1976b). Chronic exposure to benzene vapors can produce reduced leukocyte, platelet, and red blood cell counts (EPA, 1985a). Inhalation experiments conducted in rats, mice, guinea pigs, and rabbits suggest that benzene is not teratogenic at doses that are fetotoxic and embryolethal (IARC, 1982a). It has been shown to be embryo/fetotoxic at maternally toxic dose levels and it is a potent inhibitor of growth and development <u>in utero</u> (EPA,

1985a). Increased incidences of fetal resorptions, skeletal variations and altered fetal hematopoiesis have been reported (ATSDR, 1987a). Animal experiments in rats, guinea pigs, and rabbits suggest that exposure to benzene vapors may damage the testes of adult males (IARC, 1982a).

Epidemiological studies in occupational settings have described a causal relationship between exposure to benzene by inhalation (either alone or in combination with other chemicals) and leukemia in humans (IARC, 1982a). Benzene has also been shown to induce both solid tumors and leukemias and lymphomas in rats and mice exposed by gavage (Maltoni et al., 1985a; NTP, 1986a) and leukemias and lymphomas in mice exposed by inhalation (Snyder et al., 1980; Cronkite et al., 1985). EPA (1986a) classified benzene in Group A – Human Carcinogen based on adequate evidence of carcinogenicity from epidemiological studies. EPA (1989a) derived both oral and inhalation cancer potency factors of 2.9×10^{-2} $(mg/kg/day)^{-1}$ for benzene. This value was based on several studies in which increased incidences of nonlymphocytic leukemia were observed in humans occupationally exposed to benzene principally by inhalation (Rinsky, 1981; Ott, 1978; Wong et al., 1983).

3.2.3 BENZENE HEXACHLORIDE (BHC)

Technical-grade benzene hexachloride (also known as BHC or hexachlorocyclohexane) is composed mainly of the alpha (55-80%), beta (5-14%), gamma (8-15%), delta (2-16%), and epsilon (1-5%) isomers. BHC is absorbed by humans and animals following oral, inhalation, and dermal exposures (EPA, 1985u; Hayes, 1982). Absorption of the various isomers of BHC following ingestion is greater than 90% of the administered dose (Albro and Thomas, 1974). The alpha, beta, and delta-isomers of BHC primarily act as depressants of the central nervous system producing symptoms of tremors, prostration, and flaccidity of the entire musculature. Lindane (gamma-BHC) is a stimulant causing hyperexcitability, convulsions, headache, and nausea following exposure by pulmonary or ingestion routes (Hayes, 1982; EPA, 1978; Deichmann, 1981). Mental and motor retardation have been reported following dermal contact with lindane (Deichmann, 1981). All the isomers induce hepatic

enzymes (Hayes, 1982). Various reproductive and developmental effects from exposure to beta- and gamma-BHC have been demonstrated in rodents (Hayes, 1982; EPA, 1985u).

Hepatocellular tumors have been observed in mice exposed to alpha- and beta-BHC in the diet (EPA, 1989a). The most tumorigenic isomer is alpha-BHC, followed by the gamma-, beta-, delta-, and epsilon-isomers (Hayes, 1982; EPA, 1985u; EPA, 1989a). EPA (1989a) classified both alpha-BHC and technical-grade BHC in Group B2 – Probable Human Carcinogens, and beta-BHC in Group C–Possible Human Carcinogen. The weight of evidence classification for lindane (gamma-BHC) is currently under review by EPA (1989a), although EPA (1984a) has previously classified lindane in Group B2. EPA (1989a) has estimated cancer potency factors for alpha-BHC, technical grade-BHC, and beta-BHC of 6.3, 1.8, and 1.8 $(mg/kg/day)^{-1}$, respectively, based on studies by Ito et al. (1973), Munir et al. (1983), and Thorpe and Walker (1973), respectively. These cancer potency factors apply to both oral and inhalation exposures and were derived based on the incidence of hepatic tumors in mice exposed chronically to BHC in the diet (EPA, 1989a). An oral cancer potency factor of 1.3 $(mg/kg/day)^{-1}$ has been derived for gamma-BHC (lindane) by EPA (1989a), based on the study by Thorpe and Walker (1973) in which mice exposed to 400 ppm gamma-BHC in the diet for 110 weeks developed hepatocellular carcinomas.

An oral RfD for gamma-BHC (lindane) of 3×10^{-4} mg/kg/day has been derived by EPA (1989a) based on an unpublished study in which rats were administered gamma-BHC in the diet for 12 weeks (Zoecon Corp., 1983). In this study, liver and kidney toxicity were observed at 20 ppm (1.55 mg/kg/day), but not at 4 ppm (0.3 mg/kg/day). An uncertainty factor of 1,000 was used to derive the RfD. This RfD is used in this assessment for all isomers of BHC.

3.2.4 BENZOIC ACID

Benzoic acid is absorbed following both oral and dermal exposure (Opdyke, 1979). Large oral doses of benzoic acid produce gastric pain, nausea, and vomiting in humans (Gosselin et al., 1984). The lowest reported

oral lethal dose in humans is 500 mg/kg body weight (Opdyke, 1979). In experimental studies with cats, oral benzoic acid doses of 0.13 to 0.30 g/kg body weight given daily for 1 to 30 days induced central nervous system disturbances; longer-term feeding of benzoic acid at daily doses of 0.2 g/kg body weight induced liver damage (Opdyke, 1979). One report indicated that benzoic acid vapors are highly toxic by inhalation (Sax, 1984); however, this report provided no data relating to exposure conditions or doses. Although benzoic acid itself has not been reported to be teratogenic, experimental animals treated with benzoic acid have demonstrated increased sensitivity to the teratogenic effects of salicylic acid and aspirin (Opdyke, 1979). Benzoic acid has tested negative for mutagenic activity in a number of assay systems. No reports were available regarding the carcinogenic potential of this compound.

Benzoic acid has been approved for use in food by the Food and Drug Administration and is considered a "generally recognized as safe" (GRAS) food additive (Opdyke, 1979). The Joint FAO/WHO Expert Committee on Food Additives (1974) has estimated an acceptable daily intake (ADI) for benzoic acid by ingestion of up to 5 mg/kg. The EPA derived an oral RfD of 4 mg/kg/day for benzoic acid based on a no-observed-adverse-effect level (NOAEL) of 312 mg/day in humans (EPA, 1989a). An uncertainty factor of 1 was used in calculating the oral RfD. Health criteria for inhalation exposure have not been developed by EPA.

3.2.5 2-BUTANONE (METHYL ETHYL KETONE)

Absorption of methyl ethyl ketone from the gastrointestinal tract and from the lungs can be inferred from systemic toxic effects observed following acute oral exposure, and acute and subchronic inhalation exposures (Lande et al., 1976). Inhaled methyl ethyl ketone produces hepatotoxicity and neurological effects in rats (Cavender et al., 1983; Takeuchi et al., 1983). Schwetz et al. (1974a) reported that rats exposed to inhaled methyl ethyl ketone at concentrations of 3,000 ppm displayed retarded fetal development and

teratogenic effects (acaudia, imperforate anus, and brachygnathia). Methyl ethyl ketone has not been adequately tested for carcinogenicity.

EPA (1989a) derived an oral RfD of 5×10^{-2} mg/kg/day for methyl ethyl ketone based on a study by LaBelle and Brieger (1955) in which no effects were observed in 25 rats exposed to 235 ppm (693 mg/m^3 or 46 mg/kg/day) methyl ethyl ketone for 7 hours/day, 5 days/week for 12 weeks. EPA (1989a) derived an inhalation RfD of 9×10^{-2} mg/kg/day. An uncertainty factor of 1,000 was used to calculate both the oral and inhalation RfDs.

3.2.6 CARBON TETRACHLORIDE

Carbon tetrachloride (CCl_4) is readily absorbed following oral and inhalation exposure (EPA, 1984b). Carbon tetrachloride, like many other chlorinated hydrocarbons, acts as a central nervous system depressant (ACGIH, 1986). The toxic effects of oral and inhalation exposure to carbon tetrachloride in humans and animals include damage to the liver, kidney and lung; the liver is the principal target organ (EPA, 1985b). In animals, acute oral administration of 100-4000 mg/kg/day produces fatty infiltration and histological alterations in the liver. High doses produce irreversible liver damage and necrosis, while the effects observed following lower doses are largely reversible (EPA, 1985b). Humans occupationally exposed to 5-15 ppm of carbon tetrachloride experienced less severe effects including biochemical alterations, nausea, headaches and in more severe cases, liver dysfunction (jaundice, enlargement and fatty infiltration) (ACGIH, 1986; EPA, 1984b). Animals chronically exposed to carbon tetrachloride exhibit effects similar to those observed from acute exposures.

Prenatal toxicity has been demonstrated in mammalian fetuses and neonates after inhalation exposure of pregnant rats (EPA, 1984b). Carbon tetrachloride has not been shown to be teratogenic (EPA, 1985b).

Carbon tetrachloride is a carcinogen in animals producing mainly hepatic neoplasms. Doses of approximately 30 mg/kg/day or greater for 6 months have

been observed to produce an increased frequency of hepatomas, hepatocellular adenomas and hepatocellular carcinomas in mice, rats and hamsters (EPA, 1985b). EPA (1989a) classified carbon tetrachloride as a Group B2 carcinogen – Probable Human Carcinogen, based on its carcinogenicity in experimental animals. EPA (1989a) derived a cancer potency factor of 1.3×10^{-1} $(mg/kg/day)^{-1}$ for inhalation and oral exposure, based on several studies in which hepatocellular carcinomas and hepatomas were observed in rats, mice, and hamsters (Della Porta et al., 1961; Edwards et al., 1942; NCI, 1976c, 1976d, 1977a).

EPA (1989a) also derived an oral RfD of 7×10^{-4} mg/kg/day based on a subchronic gavage study in which liver lesions were produced in rats receiving 10 or 33 mg/kg/day (Bruckner et al., 1986). No lesions were produced at the 1.0 mg/kg/dose level (equivalent to 0.71 mg/kg/day after conversion of the 6 day/week dosing regimen used in this study to a daily basis) and this dose was identified as the no-observed-adverse effect level (NOAEL). An uncertainty factor of 1000 was used to derive the RfD.

3.2.7 CHLORDANE

Chlordane is absorbed through the skin, lungs, and gastrointestinal tract. As with many chlorinated hydrocarbon compounds, chlordane is retained primarily in body fat. Acute intoxication due to chlordane exposure primarily involves the central nervous system. Signs include hyperexcitability, blurred vision, irritability, confusion, vomiting, and tremors (Deichmann, 1981; EPA, 1978). Chronic effects of chlordane due to ingestion are "very small" (CAST, 1976). Growth retardation and lung and liver damage have been reported in rats fed levels of 150 and 300 ppm chlordane for 2 years (Ingle, 1952).

Chlordane has been shown to produce liver tumors in mice chronically exposed to chlordane in the diet (IRDC, 1973 as reviewed by Epstein 1976; NCI, 1977d; Velsicol, 1973). EPA (1989a) classified chlordane in Group B2 – Probable Human Carcinogen on the basis of these results. Oral and inhalation cancer potency factors of 1.3 $(mg/kg/day)^{-1}$ have been derived by EPA for

The monocyclic aromatic chemicals selected as chemicals of potential concern for surface soils are:

- Benzene,
- Ethylbenzene,
- Toluene, and
- Xylenes.

Benzene, ethylbenzene, toluene, and xylenes are all associated with petroleum hydrocarbons. Ethylbenzene, toluene, and xylenes were all detected in greater than 10 percent of all surface soil samples. Toluene was the most frequently detected chemical in surface soils with a detection frequency of 88%. Benzene, though detected infrequently in surface soils, was selected based on its association with known wastes and its carcinogenic potential.

Benzoic acid was selected as a chemical of potential concern based on its high frequency of detection and its association with known wastes. Benzoic acid is associated with biological degradation of monocyclic aromatic hydrocarbons.

2-Butanone was selected as a chemical of potential concern for the WDI site based on its high detection frequency of 32%.

Pentachlorophenol was selected as a chemical of potential concern based on its carcinogenic potential and its detection in greater than 5 percent of the surface soil samples.

Polycyclic aromatic hydrocarbons (PAHs) have been selected as chemicals of potential concern for surface soils at the WDI site. PAHs are associated with petroleum wastes, individual compounds were detected in 6 to 15 percent of the surface soil samples, and several have known carcinogenic potential. PAHs will therefore be considered chemicals of potential concern in this assessment.

Methylene chloride was selected as a chemical of potential concern for surface soils based on its carcinogenic potential, its frequent detection in surface soil samples (40%), and its potential association with known wastes at the WDI facility. Although frequently detected in surface soil samples, methylene chloride was also found in laboratory blanks. Since the source of the methylene chloride is not known, it will be evaluated as a chemical of potential concern for the WDI site.

Polychlorinated biphenyls (PCBs) have been selected as chemicals of potential concern for surface soils at the WDI site. Although individual PCBs were detected infrequently (less than 5% of the total samples), PCBs are commonly found as contaminants in fuel oils and have known carcinogenic potential.

A variety of other organic chemicals were detected in surface soil samples. Acetone, bis(2-ethylhexyl)phthalate, di-n-octylphthalate, and di-n-butylphthalate were all frequently detected in soil samples; however, these chemicals were also identified in the blank samples. The data validation reports qualified these data as potentially due to laboratory contamination because concentrations found in the samples were less than 10 times the concentrations found in the blanks. Therefore, these chemicals will not be considered further in this assessment.

Soils from 0 to 20 feet

Soil samples were collected from ground surface to a maximum depth of 60 feet. The majority of subsurface soil contamination at the WDI site was detected at depths ranging from 10 to 20 feet. As discussed in Section 2.1, filling of the former waste handling areas and reservoir has resulted in an increased surface elevation of 0 to 20 feet. Since grading may occur at the site and individuals may be exposed to all soils with 0 to 20 feet, soils ranging in depth from 0 to 20 feet will be considered together in

evaluation of future exposures to on-site soils. Chemicals selected for consideration in the EA are presented below. The criteria used to selected these chemicals of concern were discussed in Section 2.4.2. Tables 2-3 and 2-4 identify the maximum depth of each compound, the geometric mean and maximum concentrations, frequency of detection, and total number of these samples.

Inorganic chemicals selected as chemicals of concern for soils 0 to 20 feet at the WDI site are:

- Antimony,
- Arsenic,
- Cadmium,
- Chromium,
- Copper,
- Lead,
- Manganese
- Mercury,
- Nickel,
- Selenium,
- Thallium, and
- Zinc.

Arsenic, cadmium, chromium, and lead were selected as chemicals of concern for all soils based on their carcinogenic and toxic potential. Chromium was assumed to be present in the hexavalent oxidation state. The maximum concentrations for these chemicals exceeded their background concentrations by at least five times. Antimony, cadmium, copper, mercury, manganese, nickel, selenium, thallium, and zinc were all selected as inorganic chemicals of concern due to comparison to background. Each of these inorganics had maximum concentrations exceeding background by at least five times. Geometric mean concentrations were all within background range.

Organic chemicals were detected in soils from 0 to 20 feet in depth. Statistical analysis of the results are presented in Table 2-4. The organic

TABLE 2-3

INORGANIC COMPOUNDS DETECTED IN SOILS
AT DEPTHS FROM 0 TO 20 FEET
WASTE DISPOSAL, INC. SITE

COMPOUND	DETECTION FREQUENCY	NUMBER OF SAMPLES	CONCENTRATION (mg/kg) GEOMETRIC MEAN	MAXIMUM	BACKGROUND (a)
Aluminum	100%	170	14,000	32,100	>100,000
Antimony	40%	170	5.2	25	<1
Arsenic	100%	170	6.5	337	6.5
Barium	100%	170	210	4,450	700
Beryllium	85%	170	54.00	1.4	2-15
Cadmium	51%	170	1.0	18.2	<1-10
Calcium	100%	170	7,140	92,400	18,000-28,000
Chromium	100%	170	26	149	50
Cobalt	100%	170	12	29	15-70
Copper	99%	170	30	721	20
Iron	100%	170	22,000	44,900	30,000
Lead	100%	170	20	2,790	15
Magnesium	100%	170	6,400	27,200	5,000-7,000
Manganese	100%	170	400	2,270	500
Mercury	70%	170	0.130	11	<0.01-0.02
Molybdenum	36%	170	1.2	33.4	<3
Nickel	100%	170	20	105	20
Potassium	100%	167	3,100	7,710	20,000
Selenium	38%	170	0.41	1.2	0.15-0.20
Silver	34%	170	0.92	4.8	<0.5-5
Sodium	84%	167	240	6,650	15,000-100,000
Thallium	49%	170	14	39	0.1-0.8
Vanadium	100%	170	42	80	150-500
Zinc	100%	170	83	490,490	74

(a) - Sources: Shacklette and Boerngen, 1984; Conner et al., 1975; Bowen, 1979.

TABLE 2-4
ORGANIC COMPOUNDS DETECTED IN SOILS (0 TO 20 FEET)
AT THE WASTE DISPOSAL, INC. SITE

Chemical	Count	Frequency of Detection	CONCENTRATION (ug/kg) Geometric Mean (a)	Maximum
ALIPHATIC HYDROCARBONS				
Carbon Tetrachloride	1	1.9%	2.0	2
bis(2-Chloroethoxy)methane	1	1.8%	20	20
Chloroform	8	3.9%	1.6	5
1,1-Dichloroethylene	3	2.1%	62	1,200
1,2-Dichloroethylene	3	2.4%	4.4	13
Hexachloroethane	1	2.9%	280	280
Methylene Chloride	42	33%	18	1,200
1,1,2,2-Tetrachloroethane	1	1.9%	28	28
Tetrachloroethylene	7	3.8%	28	43,000
1,1,1-Trichloroethane	6	3.3%	500	1,800
Trichloroethylene	4	2.8%	140	5,000
Vinyl Acetate	1	2.9%	9.0	9
Vinyl Chloride	3	3.1%	31	1,700
CHLORINATED MONOCYCLIC AROMATIC HYDROCARBONS				
Chlorobenzene	1	1.9%	70	70
1,2-Dichlorobenzene	3	2.1%	43	1,600
1,4-Dichlorobenzene	4	2.7%	170	2,400
1,2,4-Trichlorobenzene	3	2.0%	190	2,600
CHLORINATED PESTICIDES				
Aldrin	1	2.4%	23	23
BHC (delta isomer)	2	3.8%	470	980
BHC (gamma isomer)	1	1.7%	15	15
Chlordane (alpha isomer)	7	6.4%	32	860
Chlordane (gamma isomer)	5	4.7%	47	1,200
DDD	18	6.8%	83	62,000
DDE	19	6.6%	41	30,000
DDT	17	10%	82	260,000
Dieldrin	3	7.7%	63	280
Endosulfan II	1	2.6%	120	120
Endrin	2	2.1%	4.3	14
Heptachlor	1	1.8%	87	87
Heptachlor epoxide	2	4.7%	9.6	46
KETONES				
Acetone	35	26%	58	4,100
2-Butanone	63	27%	16	11,000
2-Hexanone	1	1.9%	3.0	3
Isophorone	2	2.0%	96	150
4-Methyl-2-Pentanone	7	3.6%	4.5	68

(a) Geometric mean of positively detected samples only.

TABLE 2-4 (cont'd)
ORGANIC COMPOUNDS DETECTED IN SOILS (0 TO 20 FEET)
AT THE WASTE DISPOSAL, INC. SITE

Chemical	Count	Frequency of Detection	CONCENTRATION (ug/kg) Geometric Mean (a)	Maximum
NITROGEN-CONTAINING COMPOUNDS				
4-Chloroaniline	1	2.6%	140	140
2,4-Dinitrotoluene	1	2.0%	130	130
2,6-Dinitrotoluene	1	2.0%	360	360
Nitrobenzene	1	2.0%	20	20
4-Nitrophenol	2	4.3%	1,000	1,700
N-Nitrosodiphenylamine	4	3.3%	670	1,400
ORGANIC ACIDS				
Benzoic Acid	7	5.2%	170	1,300
PHENOLS				
4-Chloro-3-methylphenol	4	2.6%	73	180
2-Methylphenol	1	2.2%	79	79
4-Methylphenol	2	2.5%	260	630
Pentachlorophenol	5	3.8%	240	320
Phenol	3	2.2%	3,300	4,800
PHTHALATES				
Bis(2-ethylhexyl)phthalate	39	18%	170	830,000
Butylbenzylphthalate	7	5.1%	300	17,000
Di-n-butylphthalate	25	11%	200	8,600
Diethylphthalate	1	3.0%	22	22
Dimethylphthalate	1	2.2%	1,000	1,000
Di-n-octylphthalate	7	8.4%	370	88,000
POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)				
Carcinogenic				
Benzo(a)anthracene	11	4.9%	260	1,500
Benzo(a)pyrene	14	5.2%	250	1,500
Benzo(b)fluoranthene	9	5.6%	310	2,200
Benzo(k)fluoranthene	5	3.8%	150	410
Chrysene	29	11%	180	7,400
Indeno(1,2,3-c,d)pyrene	3	3.2%	190	450
Noncarcinogenic				
Acenaphthene	3	3.8%	64	170
Anthracene	9	3.7%	88	16,000
Benzo(g,h,i)perylene	6	4.3%	270	660
Dibenzofuran	5	2.9%	87	1,300
Fluoranthene	24	8.8%	120	1,500
Fluorene	31	12%	190	13,000
2-Methylnaphthalene	57	21%	1,000	170,000
Naphthalene	41	15%	720	52,000
Phenanthrene	47	18%	470	44,000
Pyrene	34	13%	160	4,300

(a) Geometric mean of positively detected samples only.

TABLE 2-4 (cont'd)
ORGANIC COMPOUNDS DETECTED IN SOILS (0 TO 20 FEET)
AT THE WASTE DISPOSAL, INC. SITE

Chemical	Count	Frequency of Detection	CONCENTRATION (ug/kg) Geometric Mean	Maximum
MONOCYCLIC AROMATIC HYDROCARBONS				
Benzene	23	9.1%	110	19,000
Ethylbenzene	51	20%	160	30,000
Styrene	3	4.9%	31	650
Toluene	137	55%	77	120,000
Xylenes	36	14%	380	250,000
NITROGEN-CONTAINING COMPOUNDS				
4-Chloroaniline	1	2.6%	140	140
2,4-Dinitrotoluene	1	2.0%	130	130
2,6-Dinitrotoluene	1	2.0%	360	360
Nitrobenzene	1	2.0%	20	20
4-Nitrophenol	2	4.3%	1,000	1,700
N-Nitrosodiphenylamine	4	3.3%	670	1,400
ORGANIC ACIDS				
Benzoic Acid	7	5.2%	170	1,300
PHENOLS				
4-Chloro-3-methylphenol	4	2.6%	73	180
2-Methylphenol	1	2.2%	79	79
4-Methylphenol	2	2.5%	260	630
Pentachlorophenol	5	3.8%	240	320
Phenol	3	2.2%	3,300	4,800
PHTHALATES				
Bis(2-ethylhexyl)phthalate	39	18%	170	830,000
Butylbenzylphthalate	7	5.1%	300	17,000
Di-n-butylphthalate	25	11%	200	8,600
Diethylphthalate	1	3.0%	22	22
Dimethylphthalate	1	2.2%	1,000	1,000
Di-n-octylphthalate	7	8.4%	370	88,000
POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)				
Carcinogenic				
Benzo(a)anthracene	11	4.9%	260	1,500
Benzo(a)pyrene	14	5.2%	250	1,500
Benzo(b)fluoranthene	9	5.6%	310	2,200
Benzo(k)fluoranthene	5	3.8%	150	410
Chrysene	29	11%	180	7,400
Indeno(1,2,3-c,d)pyrene	3	3.2%	190	450
Noncarcinogenic				
Acenaphthene	3	3.8%	64	170
Anthracene	9	3.7%	88	16,000
Benzo(g,h,i)perylene	6	4.3%	270	660
Dibenzofuran	5	2.9%	87	1,300
Fluoranthene	24	8.8%	120	1,500
Fluorene	31	12%	190	13,000
2-Methylnaphthalene	57	21%	1,000	170,000
Naphthalene	41	15%	720	52,000
Phenanthrene	47	18%	470	44,000
Pyrene	34	13%	160	4,300

TABLE 2-4 (cont'd)
ORGANIC COMPOUNDS DETECTED IN SOILS (0 TO 20 FEET)
AT THE WASTE DISPOSAL SITE

Chemical	Count	Frequency of Detection	CONCENTRATION (ug/kg) Geometric Mean (a)	Maximum
MONOCYCLIC AROMATIC HYDROCARBONS				
Benzene	23	9.1%	110	19,000
Ethylbenzene	51	20%	160	30,000
Styrene	3	4.9%	31	650
Toluene	137	55%	77	120,000
Xylenes	36	14%	380	250,000
POLYCHLORINATED BIPHENYLS (PCBs)				
Aroclor-1242	1	2.1%	80	80
Aroclor-1248	2	2.1%	190	1,700
Aroclor-1254	4	4.3%	190	570
Aroclor-1260	6	4.6%	670	3,200
OTHERS				
Carbon Disulfide	3	2.3%	6.5	10

(a) Geometric mean of positively detected samples only.

compounds were grouped as described in the previous section on surface soils, with the addition of chlorinated monocyclic aromatic hydrocarbons.

Chlorinated insecticides selected as chemicals of potential concern for all soils include:

- Aldrin,
- BHC (delta and gamma isomers),
- Chlordane (alpha and gamma isomers)
- DDT, DDD, DDE,
- Dieldrin,
- Heptachlor, and
- Heptachlor epoxide.

Aldrin, BHC, chlordane (alpha and gamma isomers), heptachlor, and heptachlor epoxide, all have known carcinogenic potential and were selected on this basis. DDT, DDD, DDE, chlordane (alpha isomer), and dieldrin were all detected in greater than 5% of the samples collected, associated with Toxo Spray Dust activities, and have carcinogenic properties. In future discussions, DDT will refer to the sum of detected DDT, DDD, and DDE concentrations.

Pentachlorophenol was selected based on its carcinogenic potential.

Monocyclic aromatic hydrocarbons selected as chemicals of potential concern for subsurface soils are:

- Benzene,
- Ethylbenzene,
- Toluene, and
- Xylenes.

Benzene, toluene, and xylenes are all related to petroleum hydrocarbon waste. Ethylbenzene, toluene, and xylenes were detected in greater than 10

percent of the samples. Toluene was the most frequently detected compound at 55%. Benzene was selected as a chemical of potential concern based on its association with known wastes and its carcinogenic potential.

2-Butanone was the only ketone selected as a chemical of concern for the WDI site. Its selection was based on a high frequency of detection (27%).

Benzoic acid was selected based on its frequency of detection and its association with known wastes.

Polycyclic aromatic hydrocarbons, both carcinogenic and noncarcinogenic, were selected as chemicals of concern for all soils at the WDI site. PAHs are associated with petroleum wastes and individual compounds were detected in up to 21% of the samples.

PCBs were also selected as chemicals of potential concern for all soils. As discussed earlier, PCBs are suspected human carcinogens and are common contaminants of fuel oils; and therefore were selected as chemicals of potential concern.

The following chlorinated aliphatic hydrocarbons were selected as chemicals of potential concern for all soils:

- Carbon tetrachloride,
- Chloroform,
- Methylene chloride,
- Tetrachloroethylene,
- Trichloroethylene, and
- Vinyl chloride.

Although all these chemicals but methylene chloride were detected in less than 5% of the samples, these chemicals have carcinogenic potential and were selected on this basis. Methylene chloride was detected in 33 percent of the samples. Tetrachloroethylene (PCE), trichloroethylene (TCE), methylene

chloride, chloroform, and carbon tetrachloride were extensively used as industrial solvents and may have been disposed at WDI. Vinyl chloride is a degradation product of PCE and TCE (Vogel et al., 1987). All these chemicals were detected in subsurface gas samples as well as in soil samples and, therefore, are assumed to be related to the WDI site.

Acetone, di-n-butylphthalate, butylbenzyl phthalate, and bis(2-ethylhexyl)phthalate, were also detected in soil samples, but were eliminated from consideration as chemicals of potential concern due to their presence in laboratory and field blank samples.

2.4.2.2 Groundwater

Twenty-seven groundwater monitoring wells were installed and sampled during the RI at WDI. Two upgradient off-site wells north of the WDI site were also sampled during the RI. Organic chemical contamination was not detected in the deep well GW-30. Monitoring wells are identified in Figure 2-12. In previous sampling efforts at the WDI site, Dames and Moore installed three monitoring wells on site (Dames and Moore, 1985). No metals or priority pollutants were detected in the samples. Samples were analyzed for the complete suite of Target Compound List (TCL) chemicals which includes volatile and semi-volatile organics, pesticides, PCBs, and metals. Geometric means and maxima for all groundwater chemicals are presented in Table 2-5.

Many inorganic elements are natural constituents of groundwater and therefore, their presence in water may not be related to chemical releases from the site. It is therefore desirable to compare concentrations detected in on-site wells with concentrations detected in background wells. Two background wells (GW-01 and GW-02) were installed in the shallow aquifer during the RI. In one of these wells, GW-01, high levels of inorganic constituents were detected. This well was resampled in August 1989 and the results were significantly different than those detected in March 1989. The initial sample from GW-01 was believed to contain colloidial iron particles,

Figure 2-12
GROUNDWATER MONITORING WELL LOCATIONS
WASTE DISPOSAL INC.

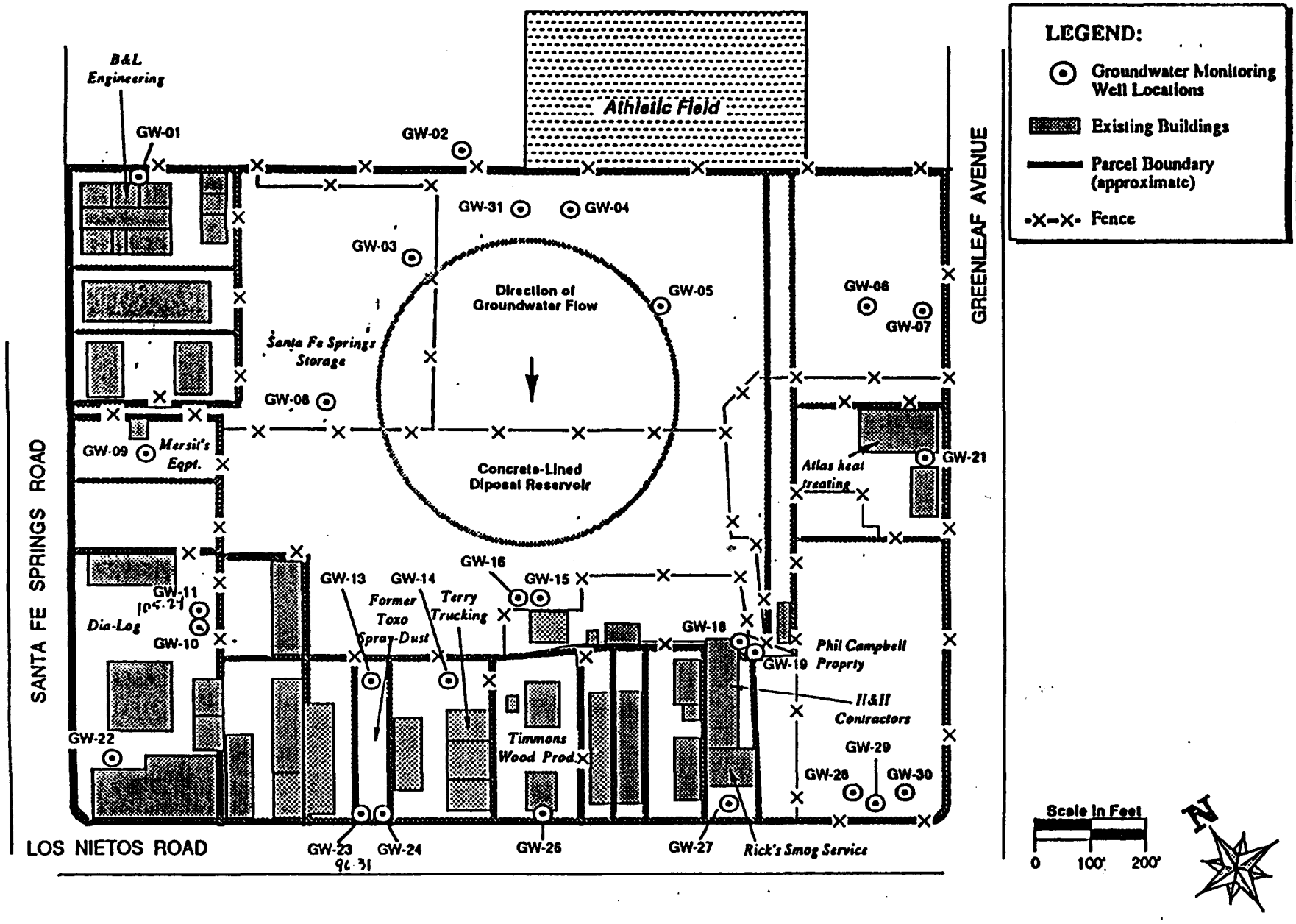


TABLE 2-5

COMPOUNDS DETECTED IN GROUNDWATER
AT THE WASTE DISPOSAL, INC. SITE

Chemical	Frequency of Detection	Number of Samples	CONCENTRATION (ug/liter) Geometric Mean (a)	CONCENTRATION (ug/liter) Maximum	Geometric Mean of Upgradient Wells (ug/liter)
INORGANICS					
Aluminum	92%	25	4.0	22,600	400
Antimony	12%	25	2.3 (b)	2.8	<2.0
Arsenic	60%	25	5.9	12	3.2
Barium	84%	25	99	252	108
Calcium	100%	25	185,000	314,000	260,000
Chromium	72%	25	12	53	8.5
Cobalt	8%	25	17 (b)	18	10.3
Copper	80%	25	17	47	19
Iron	96%	25	3,633	34,100	6,600
Lead	80%	25	4	16	6.3
Magnesium	100%	25	74,000	88,300	77,100
Manganese	100%	25	495	5,850	211
Mercury	24%	25	0.15	2.0	<0.2
Nickel	40%	25	23	45	16.3
Potassium	100%	25	5,700	9,260	6,540
Selenium	96%	25	27	67	30
Silver	8%	25	5	10	<7
Sodium	100%	25	150,000	193,000	135,000
Vanadium	36%	25	29	70	14.2
Zinc	96%	25	53	111	264
ORGANICS					
Acetone	4%	25	6.2	1100	NA
Bis(2-chloroethyl) ether	16%	25	10	590	NA
Chloroform	8%	25	2.8	9	NA
Di-n-butylphthalate	8%	25	2.0	2	NA
Diethylphthalate	4%	25	5.4	36	NA
Di-n-octylphthalate	4%	25	5.1	9	NA
Tetrachloroethylene	8%	25	2.6	11	NA
Toluene	36%	25	2.6	5.0	NA
Trichloroethylene	4%	25	2.7	18	NA

(a) - Geometric means are based on positive detects and one-half the detection limit, unless otherwise specified.
(b) - Geometric means based on positive detects only.
NA - Not analyzed for this sample.

which resulted in elevated levels of many inorganics. The concentrations detected in GW-02 during the March 1989 sampling will be used along with the August 1989 data for GW-01 as typical background concentrations of inorganics for the region.

In comparison to the upgradient wells, on-site groundwater inorganic concentrations varied. Aluminum, barium, beryllium, calcium, copper, iron, lead, magnesium, mercury, potassium, selenium, silver all had geometric mean concentrations less than or equal to, and maximum concentrations greater than, those detected in the upgradient wells. Zinc was the only inorganic which did not exceed the upgradient concentration in either geometric mean or maximum concentration. Antimony, arsenic, chromium, cobalt, manganese, nickel, sodium, and vanadium exceeded their upgradient concentration in both geometric mean and maximum concentrations.

Inorganic chemicals selected as chemicals of concern for groundwater at the WDI site are:

- Arsenic,
- Lead,
- Manganese, and
- Mercury.

Arsenic and lead were detected frequently in groundwater samples, have known human toxicity and were detected in concentrations above the upgradient levels. Arsenic and lead are also associated with petroleum wastes, especially drilling muds (Saxena, 1986). Therefore, arsenic and lead will be evaluated as chemicals of potential concern for groundwater.

Manganese was detected in all groundwater samples. Concentrations of manganese on the WDI site were above concentrations in both GW-01 and GW-02. Therefore, manganese will be considered a chemical of potential concern for groundwater.

Mercury was detected in 6 of 24 groundwater samples; mercury was not found in either of the background samples. Mercury is associated with oil field wastes (Saxena, 1986) and will be considered a chemical of potential concern.

Other inorganic compounds detected in groundwater include aluminum, antimony, barium, chromium, calcium, cobalt, copper, iron, magnesium, nickel, potassium, selenium, silver, sodium, vanadium, and zinc. Aluminum, calcium, magnesium, iron, potassium, and sodium were eliminated as contaminants of concern due to low toxicity (NAS, 1976a). Antimony, barium, chromium, cobalt, copper, nickel, selenium, vanadium, and zinc, were all detected below or at concentrations less than two times background concentrations for these constituents. These constituents will not be considered further in this assessment.

Few organic chemicals have been detected in groundwater underlying the WDI site. No organic chemicals were detected in the background wells.

Organic chemicals selected as chemicals of potential concern for groundwater at the WDI site are:

- Chloroform,
- Tetrachloroethylene,
- Toluene, and
- Trichloroethylene.

Toluene was detected in 36 percent of the groundwater samples collected and is associated with petroleum wastes. Chloroform, tetrachloroethylene, and trichloroethylene were detected infrequently (4-8%) but have known carcinogenic potential, and were therefore selected as chemicals of concern.

Acetone, bis(2-chloroethyl)ether, di-n-butylphthalate, di-n-octylphthalate, and diethylphthalate were detected infrequently at low

concentrations and are common laboratory contaminants; therefore, these will not be considered chemicals of concern.

2.4.2.3 Subsurface Gas

Subsurface gas wells were installed at 26 locations on the WDI site (Figure 2-13). Eighteen subsurface gas wells were installed adjacent to the outermost boundaries of each known or suspected waste handling area to assess the generation and migration of gas from the disposed wastes or biological breakdown. Two of these wells (VW-01, VW-09) were immediately adjacent to the border with St. Paul's High School. One subsurface gas well (VW-09) was installed in the center of the reservoir. Seven subsurface gas wells were installed in areas where no previous information on potential subsurface gas generation was available. The depth of these wells ranged from 34 to 40 feet. Samples from these gas wells were collected in stainless steel canisters and analyzed for ten volatile organic chemicals using California Air Resources Board (ARB) guidelines Method 102 and 103 (EBASCO, 1988a). Duplicate samples were collected at gas wells VW-09 and VW-13; two blank samples were also submitted for analysis. Except for methylene chloride, all analytes were detected in at least one sample. Table 2-6 presents the frequency of detection and the geometric mean and maximum concentration data for subsurface gas contaminants.

Prior to remedial investigation sampling, Dames and Moore installed a total of seven vapor probes at the Campbell property to assess contaminant concentration and migration as well as to estimate the width and depth of the waste handling areas. Methane and nonmethane hydrocarbons were detected in the samples from these probes; no further chemical speciation was performed (EBASCO, 1989a).

chlordane based on the significantly increased incidence of hepatocellular tumors observed in mice in the Velsicol (1973) study.

An oral RfD of 6×10^{-5} mg/kg/day has also been derived by EPA (1989a) based on a chronic dietary study in which rats were fed 0, 1, or 25 ppm chlordane in the diet for 130 weeks (Velsicol, 1973). Liver necrosis was observed in rats fed 1 ppm and this level was determined to be the LOAEL in this study. An uncertainty factor of 1,000 was used to derive the RfD.

3.2.8 CHLOROFORM

Chloroform is rapidly absorbed following oral and inhalation exposure. Acute exposure to high concentrations of chloroform in humans may result in death due to cardiac arrest or as a result of liver and/or kidney damage. Chronic exposure to lower concentrations may lead to hepatic, renal, and cardiac effects, and central nervous system depression (EPA, 1984c, 1985c). Chloroform has been reported to induce adverse reproductive effects in experimental animals. Adverse effects on pregnancy maintenance, delayed fetal development, and retarded fetal skeletal development have been reported in rats (Schwetz et al., 1974b; Thompson et al., 1974), mice (Burkhalter and Balster, 1979; Murray et al., 1979), and rabbits (Thompson et al., 1974).

Small increases in bladder, colon, and rectal cancers have been observed in several epidemiologic studies of human populations exposed to chlorinated drinking water (EPA, 1985c). However, since these cases involved simultaneous exposures to several potentially carcinogenic compounds, it is impossible to determine whether chloroform itself causes cancer in humans. In laboratory experiments, chloroform has been reported to cause an increase in kidney tumors in rats and mice, and liver carcinomas in mice (EPA, 1985c).

EPA (1989a) classified chloroform in Group B2 – Probable Human Carcinogen, on the basis of the evidence from animal studies. EPA (1989a) derived an oral cancer potency factor of 6.1×10^{-3} $(mg/kg/day)^{-1}$, based on a study in which kidney tumors were induced in rats exposed to 19, 38, 81, or

160 mg/kg/day in the drinking water (Jorgenson et al., 1985). EPA (1989a) derived an inhalation cancer potency factor of 8.1×10^{-2} $(mg/kg/day)^{-1}$ based on a study in which mice were exposed by gavage to average chloroform concentrations of 138 or 277 mg/kg/day for males and 238 or 477 mg/kg/day for females (NCI, 1976c).

EPA (1989a) also derived an RfD of 0.01 mg/kg/day for oral exposure. This value is based on a study in which dogs were exposed to chloroform in a toothpaste base at doses of 13 and 26 mg/kg/day for up to 7.5 years (Heywood et al., 1979). Liver lesions were produced at both dose levels and the low dose was identified as the lowest-observed-adverse effect level (LOAEL) in this study. An uncertainty factor of 1000 was applied to the LOAEL and the RfD of 0.01 mg/kg/day was derived.

3.2.9 DDD, DDE, DDT

DDT and DDE are absorbed through the skin and gastrointestinal tract in humans (EPA, 1984d). In humans, DDT and its metabolites are stored primarily in adipose tissue; storage of DDT in human tissues can last up to 20 years and tissue storage of DDE can last for the lifetime of the individual (NIOSH, 1978b). Acute oral exposure to DDT in humans and animals causes dizziness, confusion, tremors, convulsions, and paraesthesia of the extremities. Allergic reactions in humans following dermal exposure to DDT have also been reported (EPA, 1980a). Long-term occupational exposure to DDT and DDE results in increased activity of hepatic microsomal enzymes, increased serum concentrations of LDH, SGOT, and cholesterol, decreased serum concentrations of creatinine phosphokinase, increased blood pressure, and increased frequency of miscarriages (NIOSH, 1978b). Liver effects, neurological effects, immunotoxicity, reduced fertility, embryotoxicity, and fetotoxicity have also been reported in animals exposed to DDT or DDE (NIOSH, 1978b; McLachlan and Dixon, 1972; Schmidt, 1973).

DDT has been shown to be carcinogenic in mice and rats at several dose levels and following several different dosing regimens. The principal site of

action in these studies was the liver, but an increased incidence of tumors of the lung and lymphatic system were reported in several investigations (NIOSH, 1978; Tomatis et al., 1974; NCI, 1978b). DDE also caused hepatocellular carcinomas in both sexes in B6C3F1 mice (NCI, 1978b). EPA (1989a) classified DDT in Group B2 – Probable Human Carcinogen by the EPA Carcinogen Assessment Group. An oral cancer potency factor of 3.4×10^{-1} $(mg/kg/day)^{-1}$, derived by EPA (1989a), is based on several studies in mice and rats that reported significantly increased incidences of both benign and malignant liver tumors (Turusov et al., 1973; Terracini et al., 1973; Thorpe and Walker, 1973; Tomatis and Turusov, 1975; Cabral et al., 1982; Rossi et al., 1977).

EPA (1989a) developed an oral RfD for DDT of 5×10^{-4} mg/kg/day based on a study in which liver lesions were observed in rats fed DDT (Lang et al., 1950); an uncertainty factor of 100 was used to derive the RfD.

3.2.10 1,2-DIBROMOETHANE (ETHYLENE DIBROMIDE, EDB)

1,2-Dibromoethane (EDB) is absorbed following oral, inhalation, and dermal exposure. Acute exposure to high concentrations may result in central nervous system depression and injury to the lungs, liver, spleen and kidneys; similar effects may be produced following chronic exposure (Rowe et al., 1952). Direct skin or eye contact with liquid EDB is irritating and may produce reversible tissue damage. EDB does not produce teratogenic effects in rats or mice (Short et al., 1978), but it has been reported to produce deleterious reproductive effects in several species. Exposure to EDB produced impaired spermatogenesis in rats (Edwards et al., 1970) and bulls (Amir, 1973). EDB is mutagenic in several test systems and it has been shown to bind to DNA, and produce unscheduled DNA synthesis and DNA strand breaks both in <u>in vivo</u> and <u>in vitro</u> test systems (EPA, 1989a).

EDB has been shown to cause increased incidences of several tumor types in rats and mice exposed by gavage or inhalation. Epidemiological studies of workers occupationally exposed to EDB have been inconclusive. Increased incidences of stomach and lung tumors were induced in mice exposed to 44 or 77

mg/kg/day by gavage for 53 weeks and observed for a lifetime (NCI, 1978c). Increased incidences of stomach, liver, and blood vessel tumors were observed in rats exposed to 27 and 29 mg/kg/day for approximately one year in the same study (NCI, 1978c). Inhalation of 10 or 40 ppm EDB 6 hours/day 5 days/week for a lifetime produced tumors of the nasal cavity, mammary gland, lung and blood vessels in mice, and tumors of the nasal cavity, mammary gland, spleen, lung and blood vessels in rats (NTP, 1982). In a second inhalation study, exposure to 20 ppm EDB 7 hours/day, 5 days/week for 18 months produced spleen, adrenal gland, mammary gland and subcutaneous tissue tumors in rats (Wong et al. 1982).

EPA (1989a) classified EDB in Group B2 – Probable Human Carcinogen, on the basis of the bioassay results in rats and mice. EPA (1989a) derived an oral cancer potency factor of 85 $mg/kg/day^{-1}$, based on the incidence of stomach tumors in male rats induced in the NCI (1978c) study. EPA (1989a) also derived an inhalation cancer potency factor of 0.76 $(mg/kg/day)^{-1}$ based on the incidence of nasal cavity tumors induced in male rats in the NTP (1982) study.

3.2.11 1,4-DICHLOROBENZENE

1,4-Dichlorobenzene is a solid used as an air deodorant and as an insecticide. EPA (1987j) reports that 100% of an oral dose and 60% of an inhalation dose are absorbed when exposure persists for longer than one to three hours (Astrand, 1975; Dallas et al., 1983). The principal toxic effects of this compound in humans and experimental animals from acute and longer-term exposure include central nervous system depression, blood dyscrasias, and lung, kidney, and liver damage (EPA, 1985t; Riley et al., 1980). In humans, pigmentation and allergic dermatitis have been reported after dermal contact (EPA, 1987j). Chromosome breaks also have been observed in exposed humans (EPA, 1987j). 1,4-Dichlorobenzene was found to cause renal adenocarcinomas in male rats and carcinomas and adenocarcinomas of the liver in female mice in a 103-week gavage study (NTP, 1986b).

EPA classified 1,4-dichlorobenzene in Group B2--Probable Human Carcinogen based on adequate evidence of carcinogenicity in animals (EPA, 1987j). An

oral cancer potency factor of 2.4×10^{-2} $(mg/kg/day)^{-1}$ has been reported by EPA (EPA, 1989d). EPA (1987j) also derived an oral reference dose (RfD) for 1,4-dichlorobenzene of 0.1 mg/kg/day based on the NTP (1986b) rat study in which a no-observed-adverse-effect level (NOAEL) of 150 mg/kg/day for renal lesions was identified. An uncertainty factor of 1,000 was used to derive the RfD. This RfD was used to develop a lifetime health advisory for 1,4-dichlorobenzene. EPA (1989d) developed an inhalation RfD of 7.0×10^{-1} mg/m^3 (2×10^{-1} mg/kg/day based on adults) based on the Riley et al. (1980) study in which rats exposed to 75 ppm (454.6 mg/m^3) for 76 weeks exhibited liver and kidney effects. An uncertainty factor of 100 was used to derive the RfD.

3.2.12 1,2-DICHLOROETHANE

1,2-Dichloroethane is absorbed rapidly and completely following oral, dermal and inhalation exposure (EPA, 1985d). Effects of acute inhalation exposure in humans include irritation of mucous membranes in the respiratory tract and central nervous system depression (EPA, 1985d). Death may occur as a result of respiratory and circulatory failure. Pathological examinations typically show congestion, degeneration, necrosis and hemorrhagic lesions of the respiratory and gastrointestinal tracts, liver, kidney, spleen and lungs (EPA, 1985d). Adverse effects caused by less extreme exposures are generally associated with the gastrointestinal and nervous systems. Occupational exposure to high levels of 1,2-dichloroethane vapors results in anorexia, nausea, vomiting, fatigue, nervousness, epigastric pain, irritation of the eyes and respiratory tract, and gastrointestinal, liver, and gallbladder disease (EPA, 1985d). Chronic studies in animals also have revealed toxic effects following inhalation exposure including degeneration of the liver (EPA, 1985d). Available data suggest that 1,2-dichloroethane does not adversely affect reproductive or developmental processes in experimental animals except at maternally toxic levels (EPA, 1985d).

1,2-Dichloroethane has been reported to be carcinogenic in animals. In long-term oral bioassays sponsored by the National Cancer Institute (NCI, 1978d), increased incidences of squamous-cell carcinomas of the forestomach, mammary gland adenocarcinomas, and hemangiosarcomas were observed in rats;

pulmonary adenomas, mammary adenocarcinomas and uterine endometrial tumors were observed in mice.

EPA (1989a) classified 1,2-dichloroethane in Group B2 – Probable Human Carcinogen, based on sufficient evidence for carcinogenicity in animal studies. EPA (1989a) derived an oral and inhalation cancer potency factors of 9.1×10^{-2} $(mg/kg/day)^{-1}$ based on the incidences of hemangiosarcomas induced in rats in the NCI (1978d) study discussed above.

3.2.13 DIELDRIN

Dieldrin can be absorbed by humans from the gastrointestinal tract following ingestion of the pesticide (NIOSH, 1978a), and absorbed through human skin following percutaneous exposure (Feldmann and Maibach, 1974). NIOSH (1978a) reported that another possible route of absorption by humans is through inhalation (NIOSH, 1978a). Reported effects in humans following acute exposure to dieldrin include malaise, incoordination, headache, dizziness, gastrointestinal disturbances, and major motor convulsions (NRC, 1982). Dieldrin is acutely toxic to laboratory animals by the oral, dermal, and inhalation routes. It is mildly irritating to the eye and skin. Dieldrin affects the central nervous system, producing irritability, tremors, and convulsions (Heath and Vandekar, 1964). In experimental animals chronic oral administration of dieldrin is associated with liver and kidney damage (Walker et al., 1969; Treon and Cleveland, 1955; Murphy and Korschgen, 1970). Oral administration of dieldrin is reported to result in reproductive toxicity, fetotoxicity, and teratogenicity in mice and hamsters (Diechmann, 1972; Ottolenghi et al., 1974). Dieldrin is reported to cause a significant dose-related increase in the incidence of hepatocellular carcinoma in mice exposed in the diet (NCI, 1978a; Davis and Fitzhugh, 1962).

EPA has classified dieldrin in Group B2 – Probable Human Carcinogen based on inadequate evidence of carcinogenicity from human studies and sufficient evidence of carcinogenicity from animal studies (EPA, 1989a). EPA (1989a) reported a cancer potency factor inhalation exposures based on several studies in which hepatocellular carcinomas were observed in mice administered dieldrin in the diet (Walker et al., 1972; Thorpe and Walker, 1973; NCI, 1978a;

Tennekes et al., 1981). EPA (1989a) has established an oral reference dose (RfD) of 5.0×10^{-5} mg/kg/day for dieldrin based on liver lesions observed in rats (Walker et al., 1969). The RfD was derived using a no-observed-effect level (NOEL) of 0.005 mg/kg/day and an uncertainty factor of 100.

3.2.14 ETHYLBENZENE

Ethylbenzene is primarily absorbed via inhalation and distributed throughout the body in rats; the highest levels were detected in the kidney, lung, adipose tissue, digestive tract, and liver (Chin et al., 1980). In humans, short-term inhalation exposure can result in drowsiness, fatigue, headache, and mild eye and respiratory irritation (Bardodej and Bardodejova, 1970). Human exposure to high concentrations of ethylbenzene may cause central nervous system effects (NIOSH, 1985). Eye irritation has also been observed in experimental animals exposed to ethylbenzene (EPA, 1987a). In rats, acute exposure results in systemic effects primarily of the liver and kidney; chronic oral exposure also results in adverse hepatic and renal effects including increased organ weights and cloudy swelling (Wolf et al., 1956). Ethylbenzene was not embryotoxic, teratogenic, or maternally toxic for New Zealand white rabbits; maternal toxicity was observed in rats (Hardin et al., 1981). No information on the carcinogenic potential of ethylbenzene was located in the reviewed literature.

EPA (1989a) derived an oral RfD of 0.1 mg/kg/day for ethylbenzene based on a study in which no liver or kidney toxicity was observed in rats exposed to 136 mg/kg/day (Wolf et al., 1956). An uncertainty factor of 1,000 was used to derive the reference dose.

3.2.15 HEPTACHLOR/HEPTACHLOR EPOXIDE

Heptachlor epoxide is a contaminant of the insecticide heptachlor. Heptachlor is readily absorbed from the gastrointestinal tract following oral exposure (ATSDR, 1987b). Acute symptoms due to heptachlor exposure in both humans and animals include irritability, excessive salivation, labored respiration, muscle tremors, and convulsions (EPA, 1987b). Chronic exposure of experimental animals to heptachlor has been associated with hepatic lesions

and hepatocellular carcinoma (EPA, 1987b). Chronic inhalation or dermal exposure to heptachlor has been reported to result in anemia in humans. Wang and McMahon (1979) reported a significant increase in cerebrovascular disease in workers exposed to heptachlor for over 3 months. Mestitzova (1967) reported marked reduction in the litter size of rats, as well as reduced lifespan of suckling rats born to dams administered heptachlor in the diet. Results of studies with rodents also indicate that heptachlor epoxide induces reproductive and developmental effects (EPA, 1987b).

Heptachlor has not been associated with increased tumor incidences in occupationally exposed workers (Wang and McMahon, 1979). Several cases of leukemia have been reported following chronic inhalation or dermal exposure of humans; however in many of the reported cases, individuals were exposed to other chemicals in addition to heptachlor (EPA, 1985e, 1987b). Chronic dietary exposure has been reported to produce liver tumors in mice (NCI, 1977b; Epstein, 1976).

Heptachlor and heptachlor epoxide are classified as Group B2 agents (Probable Human Carcinogens) (EPA, 1989a). This classification applies to those agents for which there is sufficient evidence of carcinogenicity in animal studies and inadequate evidence of carcinogenicity in humans. Using the geometric mean of potency factors from four separate experiments in which mice exposed to dietary concentrations of heptachlor or heptachlor epoxide exhibited hepatocellular carcinomas. EPA (1989a) estimated an oral and inhalation cancer potency factor for heptachlor of 4.5 $(mg/kg/day)^{-1}$ and for heptachlor epoxide of 9.1 $(mg/kg/day)^{-1}$. Oral reference doses (RfDs), based on chronic systemic toxicity, have also been calculated for both heptachlor and heptachlor epoxide (EPA, 1989a). Male rats fed heptachlor in the diet for two years developed increases in liver weight at doses of 5 ppm (0.25 mg/kg/day) or greater (Velsicol, 1955). The no-observed-effect-level (NOEL) was 3 ppm (0.15 mg/kg/day). Applying an uncertainty factor of 300 to the NOEL, an oral RfD for heptachlor of 5.0×10^{-4} mg/kg/day was estimated (EPA, 1989a). In another study (Dow Chemical Company, 1955), beagle dogs of both sexes fed heptachlor epoxide in their diet for 60 weeks developed increased liver-to-body weight ratios. No NOEL was determined from this study, but a lowest-observed-effect-level (LOEL) of 0.5 ppm (0.125 mg/kg/day) was

identified from the available data. An oral RfD of 1.3×10^{-5} mg/kg/day for heptachlor epoxide was estimated from these data by applying an uncertainty factor of 1000 to the LOEL (EPA, 1989a). No inhalation RfDs are available for either heptachlor or heptachlor epoxide (EPA, 1989a).

3.2.16 METHYLENE CHLORIDE

The absorption of ingested methylene chloride is virtually complete. The amount of airborne methylene chloride absorbed increases in direct proportion to its concentration in inspired air, the duration of exposure, and physical activity. Dermal absorption has not been accurately measured (EPA, 1985s). Because of methylene chloride's high solubility in water and liquids, it is probably distributed throughout all body fluids and tissues. Acute human exposure to methylene chloride may result in irritation of eyes, skin, and respiratory track; central nervous system depression; elevated carboxyhemoglobin levels; and circulatory disorders that may be fatal. Chronic exposure of animals can produce renal and hepatic toxicity (EPA, 1985s).

There have been several chronic studies in which methylene chloride was administered to experimental animals either orally or by inhalation. The inhalation studies provided clear evidence of carcinogenicity. There is only suggestive evidence of a treatment-related increase in combined hepatocellular carcinomas and neoplastic nodules provided in drinking water studies in experimental animals (EPA, 1985s, 1985v). EPA (1989a) classified methylene chloride in Group B2 -- Probable Human Carcinogen. EPA (1985v) concluded that the induction of distant site tumors from inhalation exposure and the borderline significance for induction of tumors in a drinking water study are an adequate basis for concluding that methylene chloride by considered a probable human carcinogen via ingestion as well as inhalation. EPA (1985v) derived an inhalation cancer potency factor of 1.4×10^{-2} $(mg/kg/day)^{-1}$ based on the results of a National Toxicology Program (NTP) bioassay in which rats and mice were exposed to methylene chloride by inhalation for 6 hours/day, 5 days/week for 102 weeks. Significant increases in the incidence of mammary tumors in male and female rats and lung and liver tumors in male and female mice were reported. EPA (1985v) derived an oral cancer potency factor of 7.5

x 10^{-3} $(mg/kg/day)^{-1}$ based on the results of this bioassay and on an ingestion bioassay conducted by the National Coffee Association (NCA, 1983). In the NCA study, groups of from 50 to 200 mice received methylene chloride in the drinking water and a significant increase in the incidence of hepatocellular adenomas and/or carcinomas was reported for male mice.

An oral RfD of 0.06 mg/kg/day has been developed by EPA (1989a) based on a 2-year rat drinking water bioassay (NCA, 1983) that identified NOAELS of 5.85 and 6.47 mg/kg/day for male and female rats, respectively. Liver toxicity was observed at doses of 52.58 and 58.32 mg/kg/day for males and females, respectively. An uncertainty factor of 100 was applied to derive the RfD.

3.2.17 PENTACHLOROPHENOL

Pentachlorophenol is rapidly absorbed following oral, dermal, or inhalation exposure (EPA, 1984e). The major targets of pentachlorophenol (PCP) toxicity are the liver, kidneys, and central nervous system (EPA, 1985f). Acute PCP poisoning in humans is characterized by profuse sweating, often accompanied by fever, weight loss, and gastrointestinal complaints. Liver and kidney involvement have been observed in cases of fatal poisoning (Robson et al., 1969). Liver and kidney discoloration were observed in rats fed 10 to 30 mg/kg/day of purified PCP (Schwetz et al., 1978).

Mice exposed to technical-grade or purified (EC-7) pentachlorophenol in their diet for 2 years at dose levels of 100 to 600 ppm developed carcinomas and sarcomas at a number of sites (NTP, 1988). Male mice developed adrenal gland tumors and hepatocellular tumors. Some female mice displayed blood vessel tumors in addition to the hepatocellular tumors seen in the male mice. Adrenal gland tumors were observed in male mice treated with purified (EC-7) pentachlorophenol but not in female mice treated with technical-grade pentachlorophenol. Based on this study, EPA has classified pentachlorophenol as a Group B2 carcinogen (EPA, 1989a). A cancer potency factor is currently being developed by EPA based on this study, but is not yet available for this report. The California Department of Health Services (DHS) has developed a cancer potency factor of 1.6×10^{-2} $(mg/kg/day)^{-1}$ for both oral and inhalation

routes (DHS, 1989). Since no EPA value is available, the DHS cancer potency factor will be used in this assessment.

EPA (1989a) has established an oral RfD of 0.03 mg/kg/day based on the study by Schwetz et al. (1978) discussed above. An uncertainty factor of 100 was used in deriving the RfD.

3.2.18 POLYCHLORINATED BIPHENYLS (PCBs)

PCBs are complex mixtures of chlorinated biphenyls. The commercial PCB mixtures that were manufactured in the United States were given the trade name of "Aroclor." Aroclors are distinguished by a four-digit number (for example, Aroclor 1260). The last two digits in the Aroclor 1200 series represent the average percentage by weight of chlorine in the product.

PCBs are readily absorbed through the gastrointestinal tract and somewhat less readily through the skin; PCBs are presumably readily absorbed from the lungs, but few data are available that experimentally define the extent of absorption after inhalation (EPA, 1985g). Dermatitis and chloracne (a disfiguring and long-term skin disease) have been the most prominent and consistent findings in studies of occupational exposure to PCBs. Several studies examining liver function in exposed humans have reported disturbances in blood levels of liver enzymes. Reduced birth weights, slow weight gain, reduced gestational ages, and behavioral deficits in infants were reported in a study of women who had consumed PCB-contaminated fish from Lake Michigan (EPA, 1985g). For experimental animals, reproductive, hepatic, immunotoxic, and immunosuppressive effects appear to be the most sensitive endpoints of PCB toxicity in nonrodent species, and the liver appears to be the most sensitive target organ for toxicity in rodents (EPA, 1985g).

A number of studies have suggested that PCB mixtures are capable of increasing the frequency of tumors, including liver tumors, in animals exposed to the mixtures for long periods (Kimbrough et al., 1975; NCI, 1978e; Schaeffer et al., 1984; Norback and Weltman, 1985). Several studies have also suggested that PCB mixtures can act to promote or inhibit the action of other carcinogens in rats and mice (EPA, 1985g). EPA (1984f) classified PCBs in

Group B2 – Probable Human Carcinogen based on sufficient evidence in animal bioassays and inadequate evidence from studies in humans. The EPA Carcinogen Assessment Group (EPA, 1989a) calculated an oral cancer potency factor of 7.7 $(mg/kg/day)^{-1}$ for PCBs based on the incidence of hepatocellular carcinomas and adenocarcinomas in female Sprague-Dawley rats exposed to a diet containing Aroclor 1260 as reported in a study by Norback and Weltman (1985).

3.2.19 POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

PAHs occur in the environment as complex mixtures of many components with varying noncarcinogenic and carcinogenic toxic properties and potencies. Only a few components of these mixtures have been adequately characterized, and only limited information is available on the relative potencies of different compounds. The PAHs are often separated into two categories for the purposes of risk assessment: carcinogenic and noncarcinogenic PAHs. The International Agency for Research on Cancer (IARC, 1983) in reviewing the carcinogenicity of the PAHs, indicated those for which there was sufficient, limited, inadequate, or adequate negative evidence of carcinogenicity (Table 3-3).

PAH absorption following oral exposure is inferred from the demonstrated toxicity of PAHs following ingestion (EPA, 1984g). PAH absorption following inhalation exposure is inferred from the demonstrated toxicity of PAHs following inhalation (EPA, 1984g). It has been suggested that simultaneous exposure to carcinogenic PAHs such as benzo[a]pyrene and particulate matter can increase the effective dose of the compound (ATSDR, 1987c). PAHs are also absorbed following dermal exposure (Kao et al., 1985).

Acute effects from direct contact with PAHs and related materials are limited primarily to phytotoxicity; the primary effect is dermatitis (NIOSH, 1977). PAHs have also been shown to cause cytotoxicity in rapidly proliferating cells throughout the body; the hematopoietic system, lymphoid system, and testes are frequent targets (Santodonato et al., 1981). Some of the noncarcinogenic PAHs have been shown to cause systemic toxicity but these effects are generally seen only at rather high doses (Santodonato et al., 1981). Slight morphological changes in the livers and kidneys of rats have been reported following oral exposure to acenaphthene. Oral administration of

Table 3-3

CLASSIFICATION OF PAHs ACCORDING TO
EVIDENCE FOR CARCINOGENICITY

Chemicals for which there is sufficient evidence that they are carcinogenic in animals:

Benzo(a)anthracene
Benzo(b)fluoranthene
Benzo(j)fluroanthene
Benzo(k)fluoranthene
Benzo(a)pyrene
Dibenzo(a,h)acridine
Dibenzo(a,j)acridine
Dibenzo(a,h)anthracene
7H-Dibenzo(c,g)carbazole
Dibenzo(a,e)pyrene
Dibenzo(a,h)pyrene
Dibenzo(a,i)pyrene
Dibenzo(a,l)pyrene
Indeno(1,2,3-c,d)pyrene
5-Methylchrysene

Chemicals for which there is limited evidence that they are carcinogenic in animals:

Anthranthrene
Benzo(c)acridine
Carbazole
Chrysene
Cyclopenta (c,d)pyrene
Dibenzo(a,c)anthracene
Dibenzo(a,j)anthracene
Dibenzo(a,e)fluoranthene
2-, 3-, 4-, and 6-Methylchrysene
2- and 3-Methylfluoranthene

Chemicals for which the evidence is inadequate to assess their carcinogenicity:

Benzo(a)acridine
Benzo(g,h,i)fluoranthene
Benzo(a)fluroene
Benzo(b)fluroene
Benzo(c)fluroene
Benzo(g,h,i)perylene
Benzo(c)phenanthrene
Benzo(e)pyrene
Coronene
1,4-Dimethylphenanthrene
Fluorene
1-Methylchrysene
1-Methylphenanthrene
Perylene
Phenanthrene
Triphenylene

Chemicals for which the available data provide adequate evidence that they are not carcinogenic:

Anthracene
Fluroanthene
Pyrene

SOURCE: IARC, 1983

carcinogenic PAHs only after exposure to levels well above those required to elicit a carcinogenic response.

Carcinogenic PAHs are believed to induce tumors both at the site of application and systemically. Neal and Rigdon (1967) reported that oral administration of up to 250 ppm benzo[a]pyrene for approximately 110 days induced forestomach tumors in mice. Thyssen et al. (1981) observedrespiratory tract tumors in hamsters exposed to up to 9.5 mg/m^3 benzo[a]pyrene for up to 96 weeks.

Benzo[a]pyrene is representative of the carcinogenic PAHs and is classified by EPA in Group B2 – Probable Human Carcinogen based on sufficient evidence of carcinogenicity from animal studies and inadequate evidence from epidemiological studies (EPA, 1984g). EPA (1984h) calculated a cancer potency factor of 11.5 $(mg/kg/day)^{-1}$ for oral exposure to carcinogenic PAHs (specifically benzo[a]pyrene) based on the study by Neal and Rigdon (1967). EPA (1984h) calculated an inhalation cancer potency factor of 6.1 $(mg/kg/day)^{-1}$ for benzo(a)pyrene based on the study by Thyssen et al. (1981). These potency factors are currently under review based on a reevaluation of the data.

EPA's Environmental Criteria Assessment Office developed an oral RfD for chronic exposure to the noncarcinogenic PAH naphthalene of 4.1×10^{-1} mg/kg/day based on the development of ocular lesions in rats (Schmahl, 1955, as cited in EPA, 1986e) and epidemiological data on occupationally-exposed coke oven workers. An uncertainty factor of 100 was applied to the animal data in the development of the reference dose.

3.2.20 TETRACHLOROETHYLENE

Tetrachloroethylene is absorbed following inhalation (IARC, 1979a) and oral (EPA, 1985h,i) exposure. Tetrachloroethylene vapors and liquid also can be absorbed through the skin (EPA, 1985h,i). The principal toxic effects of tetrachloroethylene in humans and animals following acute and longer-term exposures include central nervous system depression and fatty infiltration of the liver and kidney with concomitant changes in serum enzyme activity levels

indicative of tissue damage (EPA, 1985h,i). Humans exposed to doses of between 135 and 1,018 mg/m^3 for 5 weeks develop central nervous system effects, such as lassitude and signs of inebriation (Stewart et al., 1974). The offspring of female rats and mice exposed to high concentrations of tetrachloroethylene for 7 hours daily on days 6-15 of gestation developed toxic effects, including a decrease in fetal body weight in mice and a small but significant increase in fetal resorption in rats (Schwetz et al., 1975). Mice also exhibited developmental effects, including subcutaneous edema and delayed ossification of skull bones and sternebrae (Schwetz et al., 1975).

In a National Cancer Institute bioassay (NCI, 1977c), a high incidence of hepatocellular carcinoma was observed in both sexes of mice administered tetrachloroethylene in corn oil by gavage 5 days per week for 78 weeks. Increased incidences of mononuclear cell leukemia and renal adenomas and carcinomas (combined) have also been observed in long term bioassays in which rats were exposed to tetrachloroethylene by inhalation (NTP, 1986d).

EPA (1989a) classified tetrachloroethylene as a Group B2 carcinogen – Probable Human Carcinogen, on the basis of these studies. EPA (1985i) derived an oral cancer potency factor of 5.1×10^{-2} (mg/kg/day)$^{-1}$ based on the liver tumors observed in the NCI (1977c) gavage bioassay for mice. The inhalation cancer potency factor for tetrachloroethylene of 3.3×10^{-3} (mg/kg/day)$^{-1}$ is based on the more recent NTP (1986d) inhalation bioassay (EPA, 1988b).

EPA (1988a) also has derived an oral RfD for tetrachloroethylene based on a study by Buben and O'Flaherty (1985). In this study, liver weight to body weight ratios were significantly increased in mice and rats treated with 71 mg/kg/day tetrachloroethylene in corn oil but not in animals treated with 14 mg/kg/day. Using a NOAEL of 14 mg/kg/day and applying an uncertainty factor of 1,000, an oral RfD of 1×10^{-2} mg/kg/day was derived.

3.2.21 TOLUENE

Toluene is absorbed in humans following both inhalation and dermal exposure (EPA, 1985j). In humans, the primary acute effects of exposure to toluene vapor are central nervous system (CNS) depression and narcosis. These

effects occur at concentrations of ≥200 ppm (754 mg/m^3) (von Oettingen et al., 1942a,b). In experimental animals, acute oral and inhalation exposures to toluene can result in CNS depression and lesions of the lungs, liver, and kidneys (EPA, 1987c). The earliest observable sign of acute oral toxicity in animals is inhibition of the CNS, which becomes evident at approximately 2,000 mg/kg (Kimura et al., 1971). In humans, chronic exposure to toluene vapors at concentrations of approximately 200 and 800 ppm has been associated with CNS and peripheral nervous system effects, hepatomegaly, and hepatic and renal function changes (EPA, 1987c). Toxic effects following prolonged exposure of experimental animals to toluene are similar to those seen following acute exposure (Hanninen et al., 1976, von Oettingen et al., 1942a). There is some evidence that oral exposure to greater than 0.3 ml/kg toluene during gestation results in embryotoxicity in CD-1 mice (Nawrot and Staples, 1979). Inhalation exposure of up to 1,000 mg/m^3 by pregnant rats during gestation has been associated with significant increases in skeletal retardation (Hudak and Ungvary, 1978).

Toluene has not been shown to be carcinogenic. No tumors were induced in rats exposed to toluene vapors for up to 24 months (CIIT, 1980).

EPA (1989a) derived an oral RfD for toluene based on a 24-month inhalation study in which rats were exposed to concentrations as high as 300 ppm (30 mg/kg/day) (CIIT, 1980). No adverse effects were observed in any of the treated animals. Using the NOAEL of 30 mg/kg/day and an uncertainty factor of 100, an oral RfD of 0.3 mg/kg/day was derived. EPA (1989b) reported an inhalation RfD for toluene of 1 mg/kg/day also based on this CIIT study and using an uncertainty factor of 100.

3.2.22 1,1,1-TRICHLOROETHANE

Like other chlorinated aliphatic hydrocarbons, 1,1,1-trichloroethane (1,1,1-TCA, methyl chloroform) is rapidly and completely absorbed by both the oral and pulmonary routes. Absorption through the skin is slow. 1,1,1-TCA distributes throughout the body and readily crosses the blood-brain barrier (EPA, 1984i).

The most notable toxic effects of 1,1,1-TCA in humans and animals are central nervous system depression, anesthesia at very high concentrations, and impairment of coordination, equilibrium, and judgment at lower concentrations (350 ppm and above). Cardiovascular effects, including premature ventricular contractions, and decreased blood pressure, can result from exposure to high concentrations of 1,1,1-TCA. Fatty changes in rodent livers following exposure by inhalation have been reported (EPA, 1985k). No adverse reproductive effects were observed in the offspring of rats or mice exposed to 1,1,1-TCA by inhalation (Schwetz et al., 1975; York et al., 1982).

Several bioassays have investigated the carcinogenic potential of 1,1,1-TCA in experimental animals. NTP (1984) reported preliminary results of a gavage bioassay in rats and mice in which 1,1,1-TCA increased the incidence of hepatocellular carcinomas in female mice. NTP (1984) further concluded that 1,1,1-TCA was not carcinogenic for male rats, an association between administration of the compound and increased incidences of hepatocellular carcinomas in male mice was equivocal and the study was inadequate to evaluate carcinogenicity in female rats. These results have been questioned and the study is presently being audited (Birnbaum, 1986).

EPA (1989a) calculated an oral RfD of 9×10^{-2} mg/kg/day for 1,1,1-TCA based on an inhalation study by Torkelson et al. (1958) in which groups of rats, rabbits, guinea pigs, and monkeys were exposed to 1,1,1-TCA vapors. A NOAEL of 500 ppm (90 mg/kg/day) was observed in guinea pigs in this study. An uncertainty factor of 1000 was combined with the NOAEL to derive the RfD. An inhalation RfD of 0.3 mg/kg/day for 1,1,1-TCA also has been determined by EPA (1989a).

3.2.23 TRICHLOROETHYLENE

Trichloroethylene, after oral ingestion, is virtually completely absorbed. With inhalation exposure, absorption is proportional to concentration and duration of exposure. Trichloroethylene distributes among the body tissues; metabolism occurs primarily in the liver (EPA, 19851). Trichloroethylene is a central nervous system depressant from acute and chronic exposure. Oral exposures of human to 15 to 25 ml (21 to 35 grams) of

trichloroethylene resulted in vomiting and abdominal pain, followed by transient unconsciousness. Exposure to high doses can result in death due to respiratory and cardiac failure (EPA, 19851). Hepatotoxicity has been reported in human and animal studies (EPA, 19851). Transient increased liver weights resulting from exposure to trichloroethylene was reported by Kjellstrand et al. (1981). Industrial use of trichloroethylene is often associated with dermatological problems including reddening and skin burns from contact with liquid trichloroethylene and dermatitis from exposure to its vapors. These effects are usually the result of contact with concentrated solvent, however, and no effects have been reported after exposure to trichloroethylene in dilute, aqueous solutions (EPA, 19851).

Trichloroethylene has been observed to induce increased incidences of liver tumors in mice (NCI, 1976d; NTP, 1983) and kidney tumors in male rats (NTP, 1983) following gavage exposure. Inhalation exposure has been shown to produce lung tumors in mice (Fukuda et al., 1983). EPA (1989a) classified trichloroethylene in Group B2 – Probable Human Carcinogen based on inadequate evidence in humans and sufficient evidence of carcinogenicity from animal studies. EPA (1989b) derived an oral cancer potency factor of 1.1×10^{-2} $(mg/kg/day)^{-1}$ and an inhalation cancer potency factor of 1.3×10^{-2} $(mg/kg/day)^{-1}$ based on the mouse liver tumor data in the NCI (1976d) and NTP (1983) gavage studies.

The EPA Office of Drinking Water (EPA, 1987d,e, 1988c) developed an oral RfD of 7.4×10^{-3} mg/kg/day for trichloroethylene based on a study by Kimmerle and Eben (1973) in which increased liver weights were observed in rats exposed to 55 ppm trichloroethylene, 5 days/week, for 14 weeks.

3.2.24 VINYL CHLORIDE

Vinyl chloride is rapidly absorbed in rats following ingestion and inhalation exposure. Dermal absorption of vinyl chloride is minor (EPA, 1985m). Absorbed vinyl chloride is distributed primarily to the liver and kidney, with lower levels found in muscle, lung, fat, spleen, and brain.

At high inhalation exposure levels, workers have experienced dizziness, headaches, euphoria, and narcosis. In experimental animals, inhalation exposure to high levels of vinyl chloride can induce narcosis and death. Lower doses result in ataxia, narcosis, congestion, and edema of the lungs and hyperemia in the liver (EPA, 1985m). Chronic inhalation exposure of workers to vinyl chloride is associated with hepatotoxicity, central nervous system disturbances, pulmonary insufficiency, cardiovascular toxicity, gastrointestinal toxicity, and acro-osteolysis (EPA, 1985m). Chronic studies of experimental animals exposed to vinyl chloride by inhalation or ingestion report effects involving the liver, spleen, kidneys, hematopoietic system, and skeletal system (EPA, 1984j).

Vinyl chloride has been demonstrated to be carcinogenic in humans and laboratory animals. Occupational exposure to vinyl chloride has been associated with an increased incidence of hepatic angiosarcoma. Vinyl chloride exposure has also been implicated in brain, lung, and hemolymphopoietic cancers in humans. Animal studies in several species support the findings of epidemiological studies. Chronic inhalation and ingestion of vinyl chloride has induced cancer in the liver and in other tissues in rats and mice (IARC, 1979b). Feron et al. (1981) fed rats vinyl chloride in the diet and found that levels as low as 1.7 and 5 mg/kg/day over their lifespan induced hepatocellular carcinoma and liver angiosarcomas, as well as other adverse hepatic effects.

EPA has classified vinyl chloride in Group A – Human Carcinogen based on adequate evidence of carcinogenicity from epidemiological studies (EPA, 1984j). EPA (1984j) reported an oral cancer potency factor of 2.3 $(mg/kg/day)^{-1}$ for vinyl chloride based on the long-term ingestion study in rats (Feron et al., 1981). Vinyl chloride doses in the experiment ranged from 0 to 14 mg/kg/day throughout the lifetime of the animals. Terminal sacrifices were made at week 135 for males and week 144 for females. A significant dose-related increase in the incidence of hepatocellular carcinoma and hepatic angiosarcoma was observed in both male and females. EPA (1989a) has also calculated an inhalation cancer potency factor for vinyl chloride of 2.95×10^{-1} $(mg/kg/day)^{-1}$.

3.2.25 XYLENES

Metabolism and excretion studies suggest that orally administered xylene is nearly completely absorbed. Acute exposure to relatively high concentrations of xylene causes central nervous system depression, minor reversible effects on the liver and kidneys and can irritate the eyes, nose, and throat. Target organs of xylene include the gastrointestinal tract, blood, liver, and kidneys (NIOSH, 1985). The most common symptoms found in people occupationally exposed to xylene are headache, fatigue, lassitude, irritability, and gastrointestinal disorders, including nausea, anorexia and flatulence (ACGIH, 1986). The liver is reportedly affected by longer-term exposure to lower levels of xylene (EPA, 1984k).

The National Toxicology Program (NTP) reported that oral administration of mixed xylenes does not result in tumor formation in rats or mice (NTP, 1986c).

EPA (1984k) derived an inhalation RfD of 3×10^{-1} mg/kg/day based on an inhalation study by Jenkins et al. (1970). An oral RfD of 2 mg/kg/day was derived by EPA (1987f) based on the National Toxicology Program carcinogenesis bioassay (1986c).

INORGANIC CHEMICALS

3.2.26 ANTIMONY

Absorption of antimony via oral and inhalation exposure is low (EPA, 1980b). Humans and animals exposed orally or through inhalation to either trivalent or pentavalent forms of antimony displayed electrocardiogram (ECG) changes and myocardial lesions (EPA, 1980b). Pulmonary effects including pneumoconiosis have been observed in humans exposed by inhalation, and dermatitis has occurred in individuals exposed either orally or dermally. Oral administration of therapeutic doses in humans has been associated with nausea, vomiting, and hepatic necrosis (EPA, 1980b). A single report (Balyaeva, 1967) noted an increase in spontaneous abortions, premature births,

and gynecological problems in 318 female workers exposed to a mixture of antimony metal, antimony trioxide, and antimony pentasulfide dusts.

EPA (1989a) derived an oral RfD of 4×10^{-4} mg/kg/day for antimony based on a chronic oral study (Schroeder et al., 1970) in which rats given the metal in drinking water had altered blood glucose and blood cholesterol levels and decreased lifespans. An uncertainty factor of 1,000 was used to derive the oral RfD.

3.2.27 ARSENIC

Soluble inorganic arsenic is rapidly and almost completely absorbed from the gastrointestinal tract in rats (Coulson et al., 1935). Estimates by Coulson et al. (1935) and Ray-Bettley and O'Shea (1975) indicate that greater than 95% of ingested inorganic arsenic is absorbed by man. Absorption of inhaled arsenic, in the form of an aerosol or a dust, is dependent upon particle size. Particles smaller than 5-7 microns in diameter may deposit deep in the lungs and be absorbed by the respiratory epithelium. Larger particles are deposited primarily in the upper airways, cleared from the lung by retrociliary action and swallowed (EPA, 19841). Once absorbed, arsenic is widely distributed. Acute exposure of humans to arsenic has been associated with gastrointestinal effects, hemolysis, and neuropathy. Chronic exposure of humans to this metal can produce toxic effects on both the peripheral and central nervous systems, keratosis, hyperpigmentation, precancerous dermal lesions, and cardiovascular damage (EPA, 1984m). Arsenic is embryotoxic, fetotoxic, and teratogenic in several animal species (EPA, 1984m).

Arsenic is a known human carcinogen. Epidemiological studies of workers in smelters and in plants manufacturing arsenical pesticides have shown that inhalation of arsenic is strongly associated with lung cancer and perhaps with hepatic angiosarcoma (EPA, 19841,m). Ingestion of arsenic has been linked to a form of skin cancer and more recently to bladder, liver, and lung cancer (Tseng et al., 1968; Chen et al., 1986).

EPA (1988d, 1989a) has classified arsenic in Group A – Human Carcinogen, and has developed inhalation and oral cancer potency factors of 50.1

$(mg/kg/day)^{-1}$ and 2.0 $(mg/kg/day)^{-1}$, respectively. The inhalation potency factor is the geometric mean value of potency factors derived from four occupational exposure studies on two different exposure populations (EPA, 19841). The oral cancer potency factor was based on an epidemiological study in Taiwan which indicated an increased incidence of skin cancer in individuals exposed to arsenic in drinking water (EPA, 19841). The increase in internal cancers recently associated with arsenic exposure is under active review by EPA. EPA is currently reviewing an oral RfD for arsenic of 1×10^{-3} mg/kg/day based on the current MCL. This RfD value will be used in the endangerment assessment.

3.2.28 CADMIUM

Gastrointestinal absorption of cadmium in humans ranges from 5-6% (EPA, 1985n). Pulmonary absorption of cadmium in humans is reported to range from 10% to 50% (DHS, 1986). Cadmium bioaccumulates in humans, particularly in the kidney and liver (EPA, 1985n,o). Chronic oral or inhalation exposure of humans to cadmium has been associated with renal dysfunction, itai-itai disease (bone damage), hypertension, anemia, endocrine alterations, and immunosuppression. Renal toxicity occurs in humans at a cadmium concentration of approximately 200 $\mu g/g$ in the renal cortex (EPA, 1985n). Cadmium is a well-documented animal teratogen (EPA, 1985n).

Epidemiological studies have demonstrated a strong association between inhalation exposure to cadmium and cancers of the lungs, kidney, and prostate (EPA, 1985o). In experimental animals, cadmium induces injection-site sarcomas and testicular tumors. Cadmium has not been shown to be carcinogenic following oral exposures in humans or animals. When administered by inhalation, cadmium chloride is a potent pulmonary carcinogen in rats. EPA (1989a) classified cadmium as a Group B1 carcinogen (Probable Human Carcinogen). This classification applies to agents for which there is limited evidence of carcinogenicity in humans from epidemiologic studies combined with sufficient evidence in experimental animals. EPA (1985n) derived an inhalation cancer potency factor of 6.1 $(mg/kg/day)^{-1}$ for cadmium based on an epidemiologic study by Thun et al. (1985).

EPA (1985n) has derived two separate RfD's using renal toxicity as an endpoint, and a safety factor of 10. The RfD associated with oral exposure to cadmium in drinking water is 5×10^{-4} mg/kg/day, and is based upon the LOAEL of .005 mg/kg identified in humans. The RfD associated with oral exposure to cadmium in food, or other nonaqueous oral exposures is 1×10^{-3} mg/kg/day.

3.2.29 CHROMIUM

Chromium is an essential micronutrient and is not toxic in trace quantities (EPA, 1980c). Chromium exists principally in the environment in two states, as chromium (III) and as chromium (VI). Following oral exposure, absorption of chromium (III) is low (<1%) while absorption of chromium (VI) is approximately 10% (ATSDR, 1987d). Acute oral exposure to high levels of soluble chromium (VI) and chromium (III) can produce kidney and liver damage; the target organs affected by chronic oral exposure remain unidentified (EPA, 1984n). Chronic inhalation exposure may cause respiratory system damage (EPA, 1984n). Certain chromium salts have been shown to be teratogenic and embryotoxic in mice and hamsters following intravenous or intraperitoneal injection (EPA, 1984n).

Epidemiological studies of worker populations have clearly established that inhaled chromium (VI) is a human carcinogen; the respiratory passages and the lungs are the target organs (EPA, 1984n). Inhalation of chromium (III) and ingestion of chromium (VI) or (III) have not been associated with carcinogenicity in humans or experimental animals (EPA, 1984n). EPA has classified inhaled chromium (VI) in Group A – Human Carcinogen (EPA, 1989a); inhaled chromium (III) and ingested chromium (III) and (VI) have not been classified with respect to carcinogenicity. EPA (1989a) developed an inhalation cancer potency factor of 41 $(mg/kg/day)^{-1}$ for chromium (VI) based on an increased incidence of lung cancer in workers exposed to chromium over a 6 year period, and followed for approximately 40 years (Mancuso, 1975).

EPA (1989a) derived an oral RfD of 5×10^{-3} mg/kg/day for chromium (VI) based on a study by MacKenzie et al. (1958) in which no observable adverse effects were observed in rats exposed to chromium (VI) in drinking water for 1 year. EPA (1989a) also developed an oral RfD of 1 mg/kg/day for chromium

(III) based on a study in which rats were exposed to chromic oxide baked in bread; no effects due to chromic oxide treatment were observed at any dose level (Ivankovic and Preussman, 1975). An uncertainty factor of 100 was used to calculate the RfD.

3.2.30 COPPER

Copper is an essential element. A daily copper intake of 2 mg is considered to be adequate for normal health and nutrition; the minimum daily requirement is 10 μg/kg (EPA, 1985p). Adverse effects in humans resulting from acute overexposure to copper by ingestion include salivation, gastrointestinal irritation, nausea, vomiting, hemorrhagic gastritis, and diarrhea (ACGIH, 1986). Dermal or ocular exposure of humans to copper salts can produce irritation (ACGIH, 1986). Acute inhalation of dusts or mists of copper salts by humans may produce irritation of the mucous membranes and pharynx, ulceration of the nasal septum, and metal fume fever. The latter condition is characterized by chills, fever, headache, and muscle pain. Limited data are available on the chronic toxicity of copper; however, chronic overexposure to copper by humans has been associated with anemia (ACGIH, 1986). Results of several animal bioassays suggest that copper compounds are not carcinogenic by oral administration; however, some copper compounds can induce injection-site tumors in mice (EPA, 1985p).

An oral RfD of 3.7×10^{-2} mg/kg/day was based on the current Federal drinking water standard of 1.3 mg/liter assuming a 70 kg person drinking 2 liters of water/day (EPA, 1987g). EPA has not determined an RfD for copper due to inadequate toxicity data. The RfD presented in this report is for purposes of estimating risks due to ingestion of copper.

3.2.31 LEAD

Absorption of lead from the gastrointestinal tract of humans is estimated at 10%-15% for adults, and up to 50% in children. For adult humans, the deposition rate of particulate airborne lead is in the respiratory track 30%-50%, and essentially all of the lead deposited is absorbed. Lead is stored in the body in bone, kidney, and liver (EPA, 1984o). The major adverse

effects in humans caused by lead include alterations in the hematopoietic and nervous systems. The toxic effects are generally related to the concentration of this metal in blood. Blood concentration levels of over 80 μg/dl in children and over 100 μg/dl in sensitive adults can cause severe, irreversible brain damage, encephalopathy, and possible death. Lower blood concentrations of lead (30-40 μg/dl) have been associated in humans with altered nerve conduction, altered testicular function, renal dysfunction, and anemia. Even lower blood lead concentrations (<10-20 μg/dl) have been associated with subtle deficits in learning ability. Lead exposure also has been associated with reproductive effects in humans including spontaneous abortions, premature delivery, and early membrane rupture; however, reliable exposure estimates are lacking in these cases. Decreased fertility, fetotoxic effects, and skeletal malformations have been observed in experimental animals exposed to lead (EPA, 1984o).

Oral ingestion of certain lead salts (lead acetate, lead phosphate, lead subacetate) has been associated with increased renal tumors in experimental animals, but doses of lead that induced kidney tumors were high and were beyond the lethal dose in humans (EPA, 1985q). EPA (1985q) classified these lead salts as Group B2 Carcinogens – Probable Human Carcinogens. This category applies to those agents for which there is sufficient evidence of carcinogenicity in animals and inadequate evidence of carcinogenicity in humans. EPA (1985r) noted that the available data provide an insufficient basis on which to regulate these compounds as human carcinogens and no cancer potency factors have been derived for lead compounds.

EPA (1989a) also considered it inappropriate to develop an RfD for inorganic lead and lead compounds, since no dose threshold can be identified for many of the health effects associated with lead intake. Previously, EPA had derived an RfD of 6×10^{-4} mg/kg/day based on the drinking water maximum contaminant level goal (MCLG) of 20 μg/L promulgated under the Safe Drinking Water Act. This value has since been withdrawn from EPA's Integrated Risk Information System (IRIS) pending re-evaluation. A new EPA-approved RfD is expected in 1989, but since it is not yet available, the old RfD (6×10^{-4} mg/kg/day) will be used in this risk assessment.

3.2.32 MANGANESE

Manganese is absorbed at low levels following oral or inhalation exposure (EPA, 1984p). Chronic oral and inhalation exposure of humans to manganese results in a condition known as manganism, a progressive neurological disease characterized by speech disturbances, tremors, and difficulties in walking. Altered hematologic parameters (hemoglobin concentrations, erythrocyte counts) have also been observed in persons exposed chronically. Manganese has not been reported to be teratogenic; however, it has been reported to cause depressed reproductive performance and reduced fertility in humans and experimental animals. There is no evidence that manganese is carcinogenic.

EPA (1984p) established an oral RfD of 2.1×10^{-1} mg/kg/day based on studies in which no adverse effects were observed in rats exposed to 1 mg/ml (21 mg/kg/day) in drinking water (Lai et al., 1982; Leung et al., 1981). EPA (1984p) also established an inhalation RfD of 3×10^{-4} mg/kg/day based on human epidemiological data which suggest that the exposure threshold for toxic effects is approximately 300 $\mu g/m^3$ (2 μg/day). Uncertainty factors of 100 were used in deriving both RfDs.

3.2.33 MERCURY

Elemental and inorganic mercury are poorly absorbed from the gastrointestinal tract (less than 15%) and easily absorbed by inhalation (approximately 80%) in humans. Organic mercury is almost completely absorbed from the gut and is assumed to be well absorbed via inhalation (EPA, 1984q). The extent of dermal absorption is not precisely known, but alkyl mercury is probably well absorbed. The toxicity of mercury depends to some extent on its chemical form. Irrespective of the chemical form, the major target organs for mercury toxicity are the central nervous system (CNS) and the kidney. Inorganic and organic mercury compounds can cause somewhat different neurotoxic effects initially, although both will elicit generally the same effects at higher doses (Hammond and Beliles, 1980). Organic mercury compounds are generally more neurotoxic than inorganic mercury.

Classical symptoms of elemental mercury vapor intoxication are mental disturbances, objective tremors, and gingivitis, which have been observed following chronic occupational exposure to average air concentrations of 0.1-0.2 mg/m^3 mercury (EPA, 1984q). The CNS appears to be the primary target of organic mercury intoxication. Miettinen (1973) estimated that an intake of 200 μg/day of organic mercury corresponded to a blood level of 200 ng/ml blood, which was estimated to be a threshold level for the development of neurological symptoms (EPA, 1984q). Clinical symptoms including paresthesia, loss of sensation in the extremities, ataxia, constriction of the visual field, and hearing impairment suggest damage to peripheral nerves, but histopathological documentation is lacking (WHO, 1976). CNS lesions similar to those in humans, proteinuria and morphological kidney changes have been reported in animals exposed to mercury (Koller, 1979; EPA, 1987h). Chronic low-dose industrial exposure has been shown to result in proteinuria. Methyl mercury does not appear to be nephrotoxic (Hammond and Beliles, 1980). Several investigators have reported embryotoxic and teratogenic effects in experimental animals treated with organic mercury. The most common findings are neurological effects, but skeletal malformations including cleft palate and jaw and facial defects have been reported in mice, hamsters and dogs. Brain damage, but not anatomical defects, has been reported in humans exposed prenatally to organic mercury (EPA, 1984q).

Limited data are available regarding the carcinogenic potential of mercury in humans or animals. Methyl mercury chloride has been shown to induce kidney tumors in mice in one test (Mitsumori et al., 1981), but other tests with organic and inorganic mercury have generally been negative. EPA has not evaluated the carcinogenic potential of mercury and no cancer potency factors have been derived for these compounds.

EPA (1986g) has derived an oral RfD for inorganic mercury of 0.002 mg/kg/day based on a study in which rats were exposed to mercury (as mercuric acetate) in the diet (Fitzhugh et al., 1950). A LOAEL of 2 mg/kg/day was identified based on the presence of morphological changes in the kidney and an uncertainty factor of 1,000 was used to derive the RfD (EPA, 1986g).

An RfD for methyl mercury of 0.0003 mg/kg/day was developed by EPA based on several studies investigating central nervous system effects in humans exposed to mercury. A blood level of 200 ng mercury/ml of blood was identified as the LOAEL from these studies. An uncertainty factor of 10 was applied in calculating the RfD. The earliest detected effects were CNS effects such as ataxia and paresthesia (EPA, 1986g).

3.2.34 SELENIUM

Selenium is an essential element in animals and probably in humans (NAS, 1980d; EPA, 1984r). Absorption of selenium from the gastrointestinal tract is generally high, but is dependent upon many factors including dietary intake level and the chemical nature of the compound. Data on other routes of absorption are limited, but absorption via the lungs has been suggested in at least one study (EPA, 1984r). Acute effects of selenium exposure following ingestion include digestive tract hemorrhage, degeneration of the myocardium, liver, kidney, and brain damage. Eye, nose, and throat irritation may also occur following inhalation exposure. Signs of chronic selenium exposure are depression, nervousness, dermatitis, and gastrointestinal disturbances. Occupational exposure to selenium has been reported to result in respiratory and gastrointestinal irritation, cold-like symptoms and metallic taste in the mouth (EPA, 1984r). Adverse reproductive effects including failure to breed and increased perinatal death have been observed in animals (EPA, 1984r). Several reports suggest that selenium may be a teratogen in humans, but this has not been conclusively proven (EPA, 1984r).

There are no epidemiological studies that suggest that selenium may be carcinogenic in humans (EPA, 1984r; IARC, 1975). However, a few studies have suggested that certain selenium compounds may cause liver tumors in laboratory animals. EPA has not classified selenium as to its carcinogenic potential and no cancer potency factors have been calculated for this compound.

EPA (1984r) calculated an oral RfD of 3×10^{-3} mg/kg/day based on an epidemiological study reported by Yang et al. (1983) in which an LOAEL of 3.2 mg/day was identified. An uncertainty factor of 15 was used to derive the RfD. EPA (1984r) also derived an inhalation RfD of 1×10^{-3} mg/kg/day based

on a study of occupationally exposed workers (Glover, 1967); an uncertainty factory of 10 was included in the calculation.

3.2.35 THALLIUM

Following acute poisoning, the highest thallium concentrations are found in the kidneys with lesser amounts found in intestines, thyroid gland, testes, pancreas, skin, bone, and spleen. Large amounts are excreted in urine within the first 24 hours following the initial exposure to thallium.

Thallium is acutely toxic regardless of the chemical form of the compound or route of administration. Since 1932, hundreds of cases of thallotoxicosis due to ingestion of thallium-based pesticides have been reported (ACGIH, 1986). Of the children poisoned by thallium ingestion who survived and were later examined, mental retardation and psychoses were the most common findings (ACGIH, 1986). The effects of thallium toxicity are similar in humans and animals. The most commonly noted response to thallium exposure is alopecia, but neurological and gastrointestinal findings are frequently found. Such effects include ataxia, lethargy, painful extremities, peripheral neuropathies, convulsions, endocrine disorders, psychoses, nausea, vomiting, and abdominal pains (Bank, 1980). Chronic studies in rats exposed to thallium in their diet for 30 days exhibited marked growth depression and a nearly complete loss of hair (Clayton and Clayton, 1984). Histological changes noted for the skin of exposed animals included atrophic hair follicles and sebaceous glands.

Exposure to thallium salts during critical developmental stages in chicks and rats has been reported to be associated with the induction of adverse developmental outcomes (Karnofsky et al., 1950). Slight kidney changes were reported in pregnant rats exposed to thallium; fetal weight reduction in pups of exposed dams was also observed (Gibson and Becker, 1970).

Thallium has not been demonstrated to be carcinogenic in humans or experimental animals and may have some antitumor activity. In a study comparing the antitumor properties of several metal salts, thallium chloride was found to increase the median survival time to greater than seven times

that of control animals; of the tumor-bearing rats, 25% were long-term survivors. No reports were found in the literature reviewed on the mutagenic activity of thallium or its salts.

EPA (1988g) developed an oral RfD for thallium of 7×10^{-5} mg/kg/day based on a study in which animals were exposed to increasing levels of thallium salts (i.e., thallous oxide, thallium acetate, thallium carbonate, thallium chloride, thallous nitrate, thallium selenite, and thallium sulfate) in the diet for 15 weeks. Alopecia and a slight increase in kidney weights were observed in the 15- and 30-ppm exposure groups; the 50-ppm group had 100% mortality by the 5th week of exposure. Using 5 ppm (0.39 mg/kg/day as thallium) as the NOEL, and an uncertainty factor of 3,000 for an animal study with small group sizes, the reference dose of 7×10^{-5} mg/kg/day was derived.

3.2.36 ZINC

Zinc is absorbed in humans following oral exposure; however, insufficient data are available to evaluate absorption following inhalation exposure (EPA, 1984x). Zinc is an essential trace element necessary for normal health and metabolism and it is nontoxic in trace quantities (Hammond and Beliles, 1980). However, overexposure to zinc has been associated with a variety of adverse effects. Chronic and subchronic inhalation exposure to zinc in humans has been associated with gastrointestinal disturbances, dermatitis, and metal fume fever, a condition characterized by fever, chills, coughing, dyspnea, and muscle pain (EPA, 1984x). Chronic oral exposure of humans to zinc may cause anemia and altered hematological parameters. Reduced body weights have been observed in rats administered zinc in the diet. There is no evidence that zinc is teratogenic or carcinogenic (EPA, 1984x).

EPA (1988a) derived an oral RfD of 2×10^{-1} mg/kg/day for zinc based on studies in which anemia and reduced blood copper were observed in humans exposed to oral zinc doses of approximately 2 mg/kg/day (Porter et al., 1977; Prasad et al., 1975). An uncertainty factor of 10 was used in developing the RfD.

4.0 HUMAN EXPOSURE ASSESSMENT

This section will address the potential pathways by which human populations could be exposed to contaminants at, or originating from, the WDI site. In identifying potential pathways of exposure, both current and likely future land-use of the site and surrounding area will be considered.

An important step in identifying exposure pathways is to consider the mechanisms by which the chemicals of potential concern at the site may migrate in the environment. Fate and transport characteristics of the chemicals of concern are presented in Section 4.1. Migration pathways are discussed in Section 4.2. In Section 4.3, potential exposure pathways are presented. In Section 4.4, exposure modeling is discussed and exposure point concentrations estimated for potential exposure pathways.

4.1 FATE AND TRANSPORT OF THE CONTAMINANTS OF CONCERN

Transport of chemicals in environmental media is a function of the physical and chemical properties of the chemical and of the environmental conditions at the site. The following section presents a general discussion of the chemical properties affecting mobility and chemical transformation, and summarizes transport processes most likely to affect the chemicals detected at the WDI site.

Water solubility is a critical property affecting the environmental transport of a chemical: highly soluble chemicals can be rapidly leached from soils or waste and are generally mobile in groundwater. For inorganic contaminants, the solubility will depend on the valence state of the element and on the chemistry of the surrounding medium.

A compound's volatilization rate from water depends on its vapor pressure and water solubility. Highly water-soluble compounds generally have lower volatilization rates from water than compounds having a low water solubility. Vapor pressure, a measure of the volatility of chemicals in their

pure state, ranges from approximately 7×10^{-9} to 7.6×10^{2} mm Hg for liquids (EPA, 1986a). The Henry's law constant, which is the ratio of a compound's vapor pressure (in atmospheres) to its solubility (in moles/m^3), is a more accurate measure of volatilization behavior than is vapor pressure for estimating releases to air from water. Compounds with Henry's Law constants greater than approximately 10^{-3} can be expected to volatilize readily from water. Those with values ranging from 10^{-3} to 10^{-5} volatilize less readily, while compounds with values less than 10^{-5} volatilize slowly (Lyman et al., 1982).

The octanol-water partition coefficient (K_{ow}) is often used to estimate the extent to which an organic chemical will partition from water into lipophilic tissues of organisms, such as fish or animal fat. Log K_{ow} values generally range from -2.5 to 10.5. Chemicals with K_{ow} less than 3 are generally considered not to concentrate in animal tissues. The organic carbon partition coefficient (K_{oc}) reflects the propensity of a compound to adsorb to the organic matter found in soil. K_{oc} values for organic chemicals range from 10^{0} to 10^{7} (log K_{oc} = 0 to 7) with higher values indicating greater adsorption potential. Chemicals with values of log K_{oc} less than 3 generally do not adsorb strongly enough to soils to affect overall leachability at normal soil organic content levels, which are generally below 1% (EPA, 1979).

For inorganic contaminants, prediction of adsorption behavior is complex; the extent of adsorption depends on the soil content of organic matter, clay, and iron and aluminum hydroxides, as well as the pH of the surrounding medium. The affinity of a chemical for soil particles is defined as the soil water distribution coefficient, K_d, and is equated to the ratio of the concentration of the chemical on the soil to the concentration in the associated interstitial water. A value of 100 or greater is indicative of strong adsorption. Another indicator of reactivity of inorganic contaminants is the redox potential (Eh) which is a measure of the ability to transfer electrons in solution or in soils.

Table 4-1

Physical/Chemical Properties of the Organic Chemicals of Potential Concern (a)

WDI SITE

Chemical	Molecular Weight (g/mol)	Solubility in water (mg/l)	Ref.	Density	Ref.	Vapor Pressure (mm HG)	Ref.	Henry's Law Constant (atm-m3 /mole)	Ref.	--PARTITION COEFFICIENTS-- Organic Carbon Coef. (log Koc)	Ref.	Octanol-Water Coef. (log Kow)	Ref.
Halogenated aliphatic hydrocarbons													
Carbon Tetrachloride	154.0	7.6E+02	M	1.59	C	9.0E+01	M	2.4E-02	D	2.04	E	2.64	M
Chloroform	119.0	8.2E+03	G	1.48	A	1.5E+02	H	3.8E-03	D	1.49	E	1.97	I
1,2-Dibromoethane (EDB)	187.9	4.3E+03	B	2.17	C	1.7E+01	B	6.7E-04	AA	1.64	E	1.76	J
1,2-Dichloroethane (EDC)	99.0	8.5E+03	L	1.26	C	6.4E+01	L	1.1E-03	D	1.15	K	1.48	L
Methylene Chloride	84.9	2.0E+04	M	1.33	L	3.6E+02	N	2.6E-03	D	0.94	K	1.30	X
Tetrachloroethylene (PCE)	165.9	1.5E+02	B	1.62	A	1.8E+01	N	2.3E-02	D	2.56	K	2.60	N
1,1,1-Trichloroethane (TCA)	133.4	1.5E+03	F	1.34	C	1.2E+02	F	2.8E-02	D	2.18	K	2.50	K
Trichloroethylene (TCE)	131.3	1.1E+03	B	1.47	A	5.8E+01	D	8.9E-03	D	2.10	K	2.38	I
Vinyl Chloride	62.5	2.7E+03	Q	0.91	C	2.7E+03	B	6.9E-01	D	1.76	E	1.38	S
Monocyclic aromatic hydrocarbons													
Benzene	78.0	1.8E+03	O	0.88	A	9.5E+01	O	5.6E-03	D	1.92	E	2.12	O
1,4-Dichlorobenzene	147.0	7.4E+01	M	1.31	F	1.2E+00	L	1.6E-03	D	3.23	K	3.39	AA
Ethylbenzene	106.2	1.5E+02	U	0.86	C	7.0E+00	U	6.4E-03	D	3.04	K	3.15	U
Toluene	92.2	5.4E+02	B	0.87	A	2.8E+01	P	6.6E-03	D	2.48	K	2.73	R
Xylenes	106.2	1.5E+02	T	0.86	A	6.5E+00	B	7.0E-03	D	2.38	E	3.26	T
Ketones													
2-Butanone (Methyl Ethyl Ketone)	72.1	2.7E+05	V	0.81	C	7.8E+01	V	5.1E-05	AA	0.65	E	0.26	V

(a) All values are at 15-20 C unless otherwise stated.

Table 4-1 (continued)

Physical/Chemical Properties of the Organic Chemicals of Potential Concern (a)

WDI SITE

Chemical	Molecular Weight (g/mol)	Solubility in water (mg/l)	Ref.	Density	Ref.	Vapor Pressure (mm HG)	Ref.	Henry's Law Constant (atm-m3 /mole)	Ref.	--PARTITION COEFFICIENTS-- Organic Carbon Coef. (log Koc)	Ref.	Octanol-Water Coef. (log Kow)	Ref.
Aromatic acids													
Benzoic Acid	122.1	2.9E+03	B	(b)	NA	1.3E-03	W	7.0E-08	AA	1.74	E	1.87	B
Pesticides													
4,4'-DDD	320.0	1.0E-01	K	(b)	NA	1.9E-06	K	8.0E-06	AA	5.89	K	6.20	K
4,4'-DDE	318.0	4.0E-02	K	(b)	NA	6.5E-06	K	6.8E-05	AA	6.64	K	7.00	K
4,4'-DDT	354.5	5.0E-03	K	(b)	NA	5.5E-06	K	5.1E-04	AA	5.39	K	6.19	K
Aldrin	365.0	1.8E-01	K	(b)	NA	6.0E-06	K	1.6E-05	AA	4.98	K	5.30	K
Chlordane	409.8	6.0E-03	X	(b)	NA	2.5E-05	Y	1.9E-03	AA	3.98	E	4.78	E
Dieldrin	381.0	2.0E-01	K	(b)	NA	1.8E-07	K	4.6E-07	AA	3.23	K	3.50	K
Heptachlor	373.5	5.6E-02	K	(b)	NA	3.0E-04	K	2.9E-03	AA	3.78	E	4.40	G
Heptachlor Epoxide	389.0	3.5E-01	K	(b)	NA	3.0E-04	K	4.4E-04	AA	2.34	K	2.70	K
Lindane (gamma-BHC)	284.8	6.0E-03	Z	(b)	NA	1.1E-05	Z	6.8E-04	AA	3.59	E	5.23	Z
Phenols													
Pentachlorophenol	266.0	1.4E+01	K	(b)	NA	1.1E-04	K	2.7E-06	AA	4.72	K	5.24	AB
Polynuclear Aromatic Hydrocarbons (PAHs) (c)													
Polychlorinated Biphenyls (PCBs) (d)													

(a) All values are at 15-20 C unless otherwise stated.

(b) Densities are not shown for chemicals that are solids at room temperature.

Table 4-1 (continued)

Physical/Chemical Properties of the Organic Chemicals of Potential Concern (a)

WDI SITE

REFERENCES FOR PHYSICAL/CHEMICAL PROPERTIES

A Weast, 1983
B Verschueren, 1983
C Windholtz, 1976
D Mackay and Shiu, 1981
E Lyman et al., 1982
F EPA, 1984i
G De Shon, 1979
H Boublik, 1984
I Hansch and Leo, 1985
J Jaber et al., 1984
K Mabey et al., 1982
L EPA, 1984s
M EPA, 1984b
N EPA, 1984t
O ATSDR, 1987a
P EPA, 1984u
Q EPA, 1984j
R Tute, 1971
S EPA, 1984j
T EPA, 1980
U EPA, 1984w
V EPA, 1984v
W Wiedemann, 1972
X Hansch and Leo, 1979.
Y Bidleman et al., 1986
Z EPA, 1984a
AA Calculated from solubility, molecular weight and vapor pressure.
AB Pierce and Victor, 1978
AC McDuffie et al., 1984

The organic chemicals of potential concern can be classified into categories according to their similarity in chemical structure or physical/chemical properties (i.e., factors that would influence mobility in the environment). The chemical categories and the chemicals of concern within each category are shown in Table 4-1.

4.1.1 ENVIRONMENTAL CHEMISTRY OF THE ORGANIC CHEMICALS OF POTENTIAL CONCERN

4.1.1.1 Halogenated Aliphatic Hydrocarbons

The halogenated aliphatic hydrocarbons detected at the WDI site are volatile, moderately to poorly water-soluble compounds. These chemicals are liquids at 20°C, with the exception of vinyl chloride, which is a gas at this temperature. These chemicals have densities greater than water; therefore, they may form a separate phase at the bottom of an aquifer if present in sufficient volume. Halogenated aliphatic hydrocarbons tend to be poorly adsorbed to soils and to be persistent in groundwater.

Transformation reactions have been reported for many of the chemicals in this class. Tetrachloroethylene in anaerobic soils has been shown to be slowly transformed via a series of biotic and abiotic reactions to trichloroethene (TCE), 1,1-dichloroethene, 1,2-dichloroethene, and vinyl chloride (Bouwer and McCarty, 1983; Vogel and McCarty, 1985; Parsons et al., 1984). Anaerobic biotransformation of 1,1,1-trichloroethane (TCA) to 1,1-dichloroethane has also been observed (Vogel et al., 1987). Little or no transformation of 1,2-dichloroethane was observed in anaerobic soils after 4 months incubation (Bouwer and McCarty, 1983). Reported transformation rates for 1,2-dibromoethane ranged from 97% in 8 weeks in soil (Castro and Belser, 1968) to no degradation in 8 weeks in an anaerobic culture (Bouwer and McCarty, 1983). Researchers have reported transformation of vinyl chloride to carbon dioxide (CO_2) by acclimated anaerobic bacteria in laboratory experiments (Vogel and McCarty, 1985). However, the frequent observation of vinyl chloride accumulation in aquifers contaminated with TCE and

tetrachloroethene appears to indicate that conditions conducive to transformation of vinyl chloride to CO_2 are not prevalent in the environment. The rate and extent of transformation reactions are highly dependent on site-specific factors such as nutrient availability and microbial composition of the soil.

The ultimate fate of halogenated aliphatic hydrocarbons in surface soils and surface water is generally volatilization to the atmosphere and subsequent photooxidation (EPA, 1979). These compounds are not significantly bioaccumulated.

4.1.1.2 Monocyclic Aromatic Hydrocarbons

Benzene, 1,4-dichlorobenzene, toluene, ethylbenzene, and xylenes were selected as chemicals of potential concern for this risk assessment. These five compounds have similar physico-chemical properties (Table 4-1), and can be grouped together in a discussion of their fate and transport in the environment. Based on their high vapor pressures and relatively low water solubilities (Verschueren, 1983), the primary fate of these monocyclic aromatic hydrocarbons in surface soils or surface water is expected to be volatilization to the atmosphere (EPA, 1979). Photooxidation in the troposphere is the dominant atmospheric fate of these compounds (EPA, 1979).

Aromatic hydrocarbons may leach from soils into groundwater. The log K_{oc} values for the five compounds range from 1.9 to 3.2, indicating that sorption to organic matter in soils or sediments may occur to a limited extent. The chemicals, except 1,4-dichlorobenzene, have a liquid density less than water, and may form a separate phase above the water table if present in sufficient quantity (e.g., as part of a gasoline plume). Based on the available groundwater data for the WDI site, two phase transport in groundwater does not appear to be occurring. Vapor-phase diffusion from groundwater may be a significant transport process in unsaturated soils.

Aromatic hydrocarbons can be biologically transformed in some soils and sediments (Barker et al., 1987); the rate and extent of transformation is highly dependent on site-specific factors such as temperature, pH, and the microbial composition of the soil. Transformation byproducts of alkyl aromatics are cresols and carbon dioxide (Barker et al., 1987; Gibson et al., 1968). Monocyclic aromatic hydrocarbons are not appreciably concentrated in plant or animal tissues (EPA, 1979).

4.1.1.3 Ketones

The ketone selected as a chemical of potential concern at WDI, 2-butanone (methyl ethyl ketone) has a low Henry's Law constant, and will therefore volatilize slowly from water or wet soil. Ketones are poorly adsorbed by soils and are highly mobile in groundwater. Ketones are readily biotransformed in soils and natural waters and eventually degrade to carbon dioxide. Based on their low K_{ow}s, the ketones would not be expected to bioaccumulate in plant or animal tissue.

4.1.1.4 Organic Acids

Benzoic acid has a pK_a of 4.19; thus, the chemical is found primarily as an anion at the pH of most natural waters and soils, which generally have pHs within the 6 to 8 range. The benzoate anion is highly soluble in water, and is therefore transported rapidly in surface waters and soils. Benzoic acid was reported not to be adsorbed to two sandy soils and two clay soils (Loekke, 1984; Bailey et al., 1968). Volatilization is not a significant fate process for the chemical (Lyman, 1982). Benzoic acid is readily biotransformed to carbon dioxide in soils and surface waters. Half-lives of less than 1 day were reported for both aerobic and anaerobic soils (Ward, 1985). Bioconcentration factors for aquatic organisms range from 14 to 2,800 (HSDB, 1989).

4.1.1.5 Organochlorine Pesticides

The organochlorine insecticides of potential concern are poorly soluble, relatively nonvolatile organics which are moderately to strongly adsorbed to soils. The environmental chemistry of this group of chemicals varies widely, therefore individual profiles for the pesticides are provided below:

- ALDRIN: Based on its low water solubility and high K_{oc}, aldrin will not leach extensively from soils with high organic content, but low levels could appear in groundwater. Aldrin has been reported to volatilize from natural waters with a half-life of days (EPA, 1979). Aldrin is biotransformed in water to dieldrin; and may also undergo phototransformation in water to the compounds photoaldrin and photodieldrin. Aldrin has been reported to bioaccumulate in aquatic organisms (EPA, 1979).

- GAMMA-BHC (GAMMA-BENZENE HEXACHLORIDE OR LINDANE): Gamma-BHC is moderately sorbed to soils, and may be leached to groundwater under conditions of high rainfall or low soil organic content (Rao and Davidson, 1982). The chemical may be transformed in soils; losses are faster under anaerobic conditions. Gamma-BHC is generally resistant to chemical or biological transformation in aquatic systems (EPA, 1979); however, it may be converted to alpha-BHC by photochemical transformation. The chemical is not extensively bioconcentrated; bioconcentration factors of 60 to 1,200 were observed in a variety of aquatic organisms (HSDB, 1989).

- CHLORDANE: Technical chlordane consists of a liquid mixture of more than 45 chlorinated compounds, which include two isomers of the chlordane molecule, heptachlor, and other constituents in varying proportions (EPA, 1979). The following discussion relates to the two chlordane isomers, which constitute approximately 40 percent of the technical chlordane mixture. The most important fate processes affecting chlordane are sorption to soils or sediments, and photolysis in surface waters. Based on its log K_{oc}, chlordane would be strongly adsorbed to soils and sediments, and would not be mobile in groundwater. Chlordane is highly persistent in soils, and is generally resistant to biodegradation. Under exposure to light, technical grade chlordane has been reported to convert to a mixture of isomers with higher toxicity and greater bioaccumulation potential (Haque, 1970). Chlordane is strongly bioaccumulated in fish (BCF = 3,600 - 19,000) and has been found to concentrate in animal fats and milk (EPA, 1979).

- DDD, DDE, DDT: DDT and DDD were in use as insecticides in the United States until the early 1970s. DDD and DDE are also contaminants and breakdown products of DDT. The dominant fate and transport

characteristics of DDT, DDD and DDE are sorption to soil materials, persistence in environmental media, and volatilization. As indicated by their high K_{oc}s, these chemicals are strongly sorbed to soils, and will generally not be leached to groundwater (EPA, 1979). In surface soils, these chemicals are likely to be transported along with soil particles by runoff or wind. In spite of their low vapor pressures, volatilization is an important process leading to eventual loss from surface water and surface soils, since these chemicals are generally not significantly degraded by chemical or biological mechanisms. Reported half-lives for biotransformation of DDT to DDE in soils range from 2 years to >15 years (HSDB, 1989). DDT is strongly bioaccumulated in both aquatic and terrestrial organisms (BCFs range from 10^3 to 10^5), and is also concentrated through the food chain.

- DIELDRIN: Dieldrin is generally immobile and persistent in soils. Volatilization from surface soils and biotransformation of dieldrin are very slow (Sandborn et al., 1977). A half-life of 7 years was reported for loss of dieldrin from soil field plots (Nash and Woolsen, 1967). Dieldrin is transformed by sunlight to photodieldrin in surface waters (EPA, 1979). Bioconcentration factors in aquatic organisms of 10^2 to 10^4 have been reported (EPA, 1979).

- HEPTACHLOR: This pesticide is a component of technical chlordane. Based on its K_{oc}, heptachlor would be moderately adsorbed to soils with a high organic content. Volatilization from surface soils is an important pathway of loss from this medium. The half-life of heptachlor in soils was reported to be 6 months (EPA, 1985e). Heptachlor is transformed by some soil organisms; transformation products include 1-hydroxychlordane and heptachlor epoxide (EPA, 1979). Heptachlor is bioconcentrated in aquatic and terrestrial organisms; reported bioconcentration factors range from 10^4 to 10^5 (EPA, 1979).

- HEPTACHLOR EPOXIDE: Heptachlor epoxide is not produced commercially, but is a product of the transformation of heptachlor in the environment. Based on its K_{oc}, heptachlor epoxide would be moderately absorbed to soils. Volatilization losses from soils of 42.5% in 11 days were observed in a laboratory study (Nash, 1983). The chemical is not readily biotransformed; reported transformation rates in soil range from no transformation in 60 days to 12% transformation to 1-exohydroxychlordane in 84 days (HSDB, 1989). Heptachlor epoxide undergoes rapid photolysis when the pure chemical is exposed to sunlight; its behavior in aqueous or soil media has not been reported. Bioconcentrations factors for aquatic organisms of approximately 10^4 have been reported (EPA, 1979), and the chemical is also concentrated in the food chain.

4.1.1.6 Pentachlorophenol (PCP)

PCP is an organic acid with a pK_a of 4.75 at 25°C; thus, its physicochemical properties depend largely on the pH of the medium in which it is found. At the environmental pH and temperature of most surface and groundwaters, PCP would be expected to occur largely as the anion, unless it is associated with a nonaqueous phase liquid or is contained within a soil pore volume with low, localized pH. Organic acids such as pentachlorophenol increase in solubility with increasing pH of the medium, as dissociation into ionic species becomes favored.

Schellenberg et al. (1984) investigated the sorption of chlorinated phenols by natural sediments and aquifer materials. The authors demonstrated that sorption of the anionic form does occur, and the degree of anionic sorption was found to be highly dependent on the organic content of the sorbent, as is sorption of the neutral form of pentachlorophenol. A K_{oc} of 32,900 for the unionized form of pentachlorophenol measured on lake sediment, river sediment, and aquifer material was reported as an average of three values. An average log K_{ow} of 5.24 (±0.14) was reported for the undissociated form. These values show that pentachlorophenol would tend to partition into hydrophobic materials (Pierce & Victor, 1978).

Biodegradation has been reported for pentachlorophenol in both soil and surface water. Pentachloroanisole has been found as a major degradation product in a surface water system (Pierce and Victor, 1978), while tetra-, tri- and di-chlorophenols have been observed as degradation products in soil studies (EPA, 1979; Mikesell and Boyd, 1988). The rate and extent of PCP transformation is highly dependent on site-specific factors such as soil moisture, soil organic content, soil oxygen content, and temperature (WHO, 1987). Transformation rates were reported to be higher in anaerobic soils, and with addition of acclimated bacteria; 55% disappearance of PCP was observed within 56 days in an anaerobic soil (Mikesell and Boyd, 1988).

Table 4-2

Selected Physical and Chemical Properties of Some Polycyclic Aromatic Hydrocarbons

Compound	Molecular Weight (g/mole)	Vapor Pressure (mm Hg) (20-25°C)	Henry's Law Constant[a]	Water Solubility (mg/liter)	Log K_{ow}[b]	BCF[c] ($atm\text{-}m^3$/mole)	Density
Naphthalene	128	0.082	4.6×10^{-4}	32	3.37	146	1.16
Phenanthrene	178	6.8×10^{-4}	1.6×10^{-4}	1.3	4.46	1,230	1.17
Anthracene	178	1.9×10^{-4}	1.4×10^{-3}	0.045	4.45	1,210	1.25
Fluoranthene	202	5.0×10^{-6}	1.0×10^{-5}	0.260	4.90	2,920	1.27
Pyrene	202	2.5×10^{-6}	5.0×10^{-6}	0.13	4.88	2,800	1.27
Benz[a]anthracene	228	2.2×10^{-8}	1.2×10^{-6}	0.0057	5.61	11,700	1.27
Chrysene	228	6.3×10^{-9}	9.5×10^{-7}	0.0020	5.61	11,700	1.27
Benzo[a]pyrene	252	5.6×10^{-9}	1.5×10^{-6}	0.0012	6.06	28,200	1.35
Benzo[g,h,i]perylene	276	1.0×10^{-10}	5.3×10^{-8}	0.00070	6.51	68,200	NA

Sources: EPA (1984k); Mabey et al., (1981); Windholz (1976). NA = not available.

[a]Calculated from water solubility, vapor pressure, and molecular weight.

[b]K_{ow} = octanol/water partition coefficient

[c]BCF = bioconcentration factor, values estimated by method of Veith et al. (1979)

Bioaccumulation of PCP is dependent on the pH of the medium; therefore, observed bioconcentration factors vary over several orders of magnitude. Bioconcentration factors in fish ranging from 6 to 1050 have been reported (WHO, 1987). Model ecosystem studies by Lu et al. (1978) indicate that PCP bioaccumulates along the food chain. Measurement of PCP residues in biota is complicated by the tendency of PCP to form protein conjugates, which are not detected by some analytical methods (Kinzell, 1981).

4.1.1.7 Polycyclic Aromatic Hydrocarbons (PAHs)

The term polycyclic aromatic hydrocarbons describes a diverse class of chemicals consisting of 2 or more fused benzene rings, which vary widely in arrangement. PAHs are chiefly formed from the combustion of organic material and are widely present in the environment due to both natural and anthropogenic activities.

The physical properties of PAHs, which affect their mobility in the environment, depend largely on the sizes of the molecules; therefore molecular weights are key properties. Table 4-2 lists physicochemical properties of selected PAHs. Vapor pressures and solubilities vary widely within the class and can be correlated with molecular weight. Volatilization may be a significant transport mechanism for a low molecular weight PAH such as naphthalene, but much less significant for the higher molecular weight compounds, such as indeno(1,2,3-c,d)pyrene.

The relatively high solubilities of the lower molecular weight PAHs, such as naphthalene, acenaphthylene, acenaphthene, and fluorene, can result in some migration into groundwater. The higher molecular weight PAHs such as benzo(a)pyrene tend to adsorb onto soil surfaces, especially if the soil is high in organic carbon content. For this reason, overland transport of PAHs adsorbed onto soil particles is a typical mechanism of migration. PAHs associated with creosote or petroleum may also migrate through the unsaturated zone or through an aquifer as a non-aqueous plume.

All of the PAHs are capable of undergoing photolytic transformations in the environment, and their degradation half-lives vary widely. Photolysis is a process that is greatly affected by site-specific variables such as intensity of sunlight, turbidity of water (if in a surface water environment), depth in surface water, or nature of the adsorbent.

Biodegradation of PAHs in the environment is extremely variable across the chemical class. The process is highly complex and depends on numerous factors such as the species of microorganisms, availability of nutrients, oxygen tension, degree of acclimation, nature of the medium, concentration of the chemical, temperature, and pH. In general, the di- and tricyclic PAHs are more readily biodegraded than the tetracyclic and higher polycyclic hydrocarbons (Tabak et al., 1981). The concentrations of the PAHs present can affect their ability to be biodegraded. Tabak et al. (1981) found that fluoranthene, pyrene, and chrysene underwent significant degradation at 5 mg/L substrate levels, but were less efficiently degraded at 10 mg/L. Coover and Sims (1987) studied the persistence of PAHs at 10°C, 20°C, and 30°C in an unacclimated agricultural sandy loam soil. The authors found that increasing the soil temperature significantly improved the rate and extent of apparent loss of low molecular weight PAHs, but had little effect on the loss of five and six-ring PAHs. Volatilization may have contributed significantly to the loss of the low molecular weight PAHs such as acenaphthene, fluorene, phenanthrene, and anthracene. Because of the numerous variables involved, different investigators have measured vastly different half-lives for biodegradability of the same material.

4.1.1.8 Polychlorinated Biphenyls (PCBs)

PCBs are complex mixtures of polychlorinated biphenyls. The commercial PCB mixtures that were manufactured in the U.S. by Monsanto Corporation were given the trade name of "Aroclor", and PCB mixtures are generally identified by reference to the most similar Aroclor mixture, even if the PCBs detected were produced by another manufacturer. Aroclors are identified by a four-digit number (for example, Aroclor 1260). The last two digits in the Aroclor

1200 series represent the average percentage by weight of chlorine in the product.

The chemical, physical and biological properties of PCBs vary widely, depending on the number of chlorine atoms and the arrangement of the chlorine atoms on the PCB molecule. There are 209 possible PCB isomers, which may have from one to 10 chlorine atoms on the molecule. Each commercial PCB mixture has a characteristic isomer distribution. Typical percentages of various chlorinated isomers in each Aroclor mixture are shown in Table 4-3. Representative physical/chemical properties for Aroclor mixtures are shown in Table 4-4.

In general, PCBs have low water solubilities, ranging from about 5 mg/L (5 ppm) for mono-chloro isomers to 1.2 ng/L (1.2 ppt) for the deca-chloro isomer (Shiu and Mackay, 1986). The chemicals also have moderate to low vapor pressures. PCBs have a strong affinity to adsorb to organic materials in soils and sediments. Less-chlorinated molecules will leach from an adsorbed mixture more rapidly than the more-chlorinated ones, leaving a residue enriched in the larger molecules. The strong sorptive tendencies of PCBs means that PCBs may be transported adsorbed to soil particles, either in overland runoff or by atmospheric transport of dust.

Organic solvents or oil can mobilize PCBs in soils, since PCBs are more soluble in these liquids than in water (Griffin and Chian, 1980). Aroclors that are liquids in their pure form may also travel through the subsurface as an oily phase, if present in large quantities (Roberts et al., 1982). Volatilization of PCBs from surface soils may be an important transport process for PCBs with fewer than four chlorines.

PCBs are relatively inert to chemical or biological transformation, and therefore are persistent in the environment. Nevertheless, due to the absence of other transformation processes affecting PCBs, biodegradation may be a significant factor in the long-term fate of PCBs from soils (ATSDR, 1987d). The relative rates of biological transformation of PCBs depend on the position

Table 4-3

Approximate Molecular Composition of PCBs
(percent)

Empirical formula	Aroclor designation						
	1016	1221	1232	1242	1248	1254	1260
$C_{12}H_{10}$	<0.1	11	<0.1	<0.1	ND	<0.1	ND
$C_{12}H_9Cl$	1	51	31	1	ND	<0.1	ND
$C_{12}H_8Cl_2$	20	32	24	16	2	0.5	ND
$C_{12}H_7Cl_3$	57	4	28	49	18	1	ND
$C_{12}H_6Cl_4$	21	2	12	25	40	21	1
$C_{12}H_5Cl_5$	1	<0.5	4	8	36	48	12
$C_{12}H_4Cl_6$	<0.1	ND	<0.1	1	4	23	38
$C_{12}H_3Cl_7$	ND	ND	ND	<0.1	ND	6	41
$C_{12}H_2Cl_8$	ND	ND	ND	ND	ND	ND	8
$C_{12}H_1Cl_9$	ND	ND	ND	ND	ND	ND	ND

Source: Hutzinger et al., 1974.

TABLE 4-4

Physical-Chemical Properties of Some Commercial PCB Mixtures

	Average MW[c] (g/mole)	Solubility (mg/L) @25°C	Ref.	Vapor Pressure torr (@25°C)	Ref.	Henry's law constant ($atm.m^3$/mole)	Ref.	log K_{ow}	Ref.
Aroclor 1016	258	0.67	H	$9.0x10^{-4}$	D	$7.6x10^{-4}$	H	5.88	G
Aroclor 1221	201	6.5	H	$1.5x10^{-2}$	H	$2.3x10^{-4}$	H	4.38[d]	H
Aroclor 1232	233	2.0	H	$4.1x10^{-3}$	H	$5.9x10^{-4}$	H	4.84[d]	H
Aroclor 1242	261	0.24[a]	A,B,C	$4.3x10^{-4}$a	A,B,C,D	$2.2x10^{-4}$a	A,B,C,E,F	5.76	C
Aroclor 1248	288	0.20	H	$1.8x10^{-4}$	D	$4.4x10^{-4}$	E	6.11	G
Aroclor 1254	327	0.041[a]	A,B,C	$4.3x10^{-5}$a	A,B,C,D	$2.0x10^{-4}$a	A,B,E,F	5.89[b]	C
Aroclor 1260	372	0.014[a]	A	$1.1x10^{-5}$a	A,D	$2.5x10^{-4}$a	A,E	6.91	G

A: Murphy et al., 1987
B: Albro and Parker, 1979
C: Rappaport and Eisenreich, 1984
D: Foreman and Bidleman, 1985
E: Buckhard et al., 1985
F: Murphy et al., 1983
G: Veith et al., 1979
H: Mackay et al., 1983 (default values)

a Values for solubility, vapor pressure, and Henry's Law constants are averages of experimental data for Aroclor mixtures with values calculated based on congener-specific properties and information on the percent composition of congeners in different Aroclor mixtures. Composition data for Aroclor mixtures taken from references B, C, and D. Congener property information from references A and D.

b Calculated based on congener-specific information for K_{ow} values and the fraction of these congeners present in each Aroclor mixture as presented in reference C.

c Varies from batch to batch of commercial mixture.

d Root mean square of range reported in reference H.

and number of chlorine atoms: in general, PCBs with one to three chlorines biodegrade relatively quickly; PCBs with four chlorines biodegrade slowly, and PCBs with five or more chlorines are resistent to biodegradation (Griffin and Chian, 1980). Pal et al. (1974) concluded that certain PCB isomers with five or more chlorines have been documented to degrade at rates up to 25% per year; however, the rates appear to be highly specific to the individual isomer. PCB degradation reactions rates also tend to be highly dependent on site-specific factors such as the oxygen content of the soil and the presence of appropriate microorganisms.

4.1.2 INORGANIC CHEMICALS OF POTENTIAL CONCERN

4.1.2.1 Antimony

Antimony (Sb) is a metallic element occurring in the earth's crust in concentrations of approximately 0.2 to 0.5 mg/kg (Arthur D. Little, 1976). It can exist in four oxidation states: +5, +3, 0, and -3. The +5 form is important only in highly oxidizing environments. The chemistry of antimony is similar to that of arsenic, which directly overlies it in the periodic table. Antimony forms trivalent and pentavalent compounds with sulfur and chloride.

Under moderately oxidizing aqueous conditions, antimony occurs primarily in the +3 oxidation state, as the hydrated trioxide $Sb_2O_3(H_2O)_n$. Salts of antimonite acid can also be formed in natural waters. The oxides and salts of antimony are moderately soluble in water; therefore, antimony is considered to be mobile in surface waters (Cotton and Wilkinson, 1982). Under conditions of high acidity and in contact with reducing agents, the poisonous gas stibine (SbH_3) may be formed. Stibine is volatile and soluble in water (5000 mg/L), but is rapidly oxidized to soluble oxides under aerobic conditions (Parris and Brinkman, 1976). Volatilization may be of limited importance in loss of antimony from surface or groundwater.

The sorptive behavior of antimony has not been well characterized; however, as with arsenic, sorption is expected to be a major factor limiting

aqueous transport. Crecelius et al. (1975) reported that antimony in the marine environment may coprecipitate with iron, manganese, or aluminum oxides, and could then be available for remobilization under reducing conditions. Very little antimony was found to be complexed with humic acids. Antimony was shown to persist in soils (Crecelius et al., 1974) and in river sediments (Maxfield et al., 1974). Baes et al. (1984) reported a soil-water distribution coefficient (K_d) of 45 for antimony.

Bioaccumulation and biotransformation of antimony compounds may be of importance in some environments. Based on analogy to arsenic, biological formation of trimethyl stibine would be expected to occur; however, this process has not been detected in environmental samples (EPA, 1979). Bioconcentration of antimony has been shown to occur in fish (BCF = 40), and invertebrates (BCF = 16000) (EPA, 1979).

4.1.2.2 Arsenic

Arsenic (As) has four stable oxidation states, +5, +3, 0 and -3. As(V) and As(III) are the most common oxidation states in aqueous environments. The two states are readily interconverted by biological and chemical redox reactions. Arsenates (As(V)) predominate in most soils, while arsenites (As(III)) may dominate in reducing environments. As(III) species are generally more mobile than As(V) in the subsurface. There is evidence that arsenic may leach into groundwater, especially from soils with low sorptive capacity (EPA, 1984m). The primary processes limiting the mobility of arsenic in soils are precipitation as metal salts, coprecipitation with iron or manganese oxides, substitution for phosphorus in minerals, and adsorption to amorphous metal oxides.

In highly reduced soils, or in the presence of certain microorganisms and fungi, arsenic can be reduced to arsine (AsH_3). Arsine can be metabolized by soil microorganisms to dimethyl arsine or trimethyl arsine, which are volatile and highly toxic compounds (Bodek et al., 1988). Loss of arsenic by volatilization has been observed in a number of soils (Cox, 1975) in

laboratory experiments and in the environment. Bioaccumulation factors for arsenic in aquatic organisms were reported to range from 5 - 6,000, and were highest at the lower trophic levels (EPA, 1979).

4.1.2.3 Cadmium

Cadmium mobility in aqueous environments is controlled by the pH of the water, through formation of hydroxides: $CdOH^{+}$, $Cd(OH)_2$, $Cd(OH)_3^{-}$, and $Cd(OH)_4^{-2}$. The dominant species below pH 8 is the hydrated divalent cation, Cd^{+2} (Pourbaix, 1963, Moore and Ramamoorthy, 1984). As the pH increases, cadmium hydroxides will form and remove some of the cadmium from solution. Under reducing conditions, and in the presence of sulfur, the relatively insoluble cadmium sulfide (CdS) will form, and will control cadmium solubility (EPA, 1979). Organic materials such as humic acids can influence the speciation of cadmium by forming cadmium-organic complexes which increase the solubility of cadmium (Gardiner, 1974).

The mobility of cadmium in soils and ground water is influenced by several processes which result in a reduction of its mobility although it should be noted that in spite of these interactions, cadmium is among the most mobile elements in the environment. Sorption processes have more of an influence on cadmium mobility than do precipitation reactions (Kabata-Pendias and Pendias, 1984). Cadmium mobility is reduced by cation exchange reactions with clays and organic matter, sorption to clay (Korte et al., 1976) and metal oxides (EPA, 1979), and complexation with organic acids at low pH (Huang et al., 1977). Sorption increases at higher pH (Frost and Griffin, 1977). To summarize, although cadmium can sorb onto soils and to a lesser extent metal oxides, clays, or carbonates, these processes are only favored under specific conditions (Bodek et al., 1988). For example, below pH 6, precipitation or sorption was not found to occur (Huang et al., 1977).

Cadmium in soil is readily taken up by plants (Chaney, 1984), and is accumulated by organisms from food or water. Bioaccumulation factors for freshwater and marine organisms range from hundreds to thousands of times the

ambient water concentrations (ATSDR, 1987e). Cadmium taken up by feed crops may be bioaccumulated by livestock and thus enter the human food chain. Biological transformation of cadmium does not appear to occur (EPA, 1979).

4.1.2.4 Chromium

Chromium (Cr) occurs in two oxidation states in aqueous systems: Cr(III) and Cr(VI). The chemistries of Cr(III) and Cr(VI) are very different. Trivalent chromium (Cr(III)) reacts with hydroxide ion in water to form insoluble chromium hydroxide, $Cr(OH)_3 \bullet H_2O_n$, which is rapidly removed from water by precipitation and sorption to soils or sediments (EPA, 1979). Hexavalent chromium (Cr(VI)) forms soluble chromate and dichromate anions which are not strongly adsorbed to soils or sediments, and are therefore mobile in the environment (EPA, 1979). Cr(VI) and Cr(III) may be interconverted in soils or surface waters under conditions which change the redox potential of the system (EPA, 1979). Cr(VI) is rapidly reduced to Cr(III) in soils having a high content of organic matter, or by contact with ferrous or sulfide ions (Bartlett and Kimble, 1976). Oxidation of Cr(III) to Cr(VI) has been shown to occur in the presence of excess manganese oxide (Bartlett and James, 1979).

Chromium is accumulated in freshwater and marine biota to levels ranging from approximately 100 to 4,000 times the concentration in water (EPA, 1979). The element can be transferred up the food chain, but does not appear to be magnified at higher trophic levels in a food chain consisting of phytoplankton, brine shrimp, post-larval fish, and mummichog clams (EPA, 1979). Chromium does not appear to undergo biological transformation reactions such as methylation, but Cr(VI) may be chemically reduced to Cr(III) upon contact with plant or animal tissue.

4.1.2.5 Copper

Copper (Cu) can be found in three oxidation states: 0, +1, and +2. Of these three, only the Cu(II) oxidation state is found in aquatic systems. In

polluted environments, copper can also form complexes with cyanide, amino acids, and humic substances. In the absence of organic complexing agents, hydrolysis and precipitation dominate copper's chemistry in aqueous environments.

The interactions of copper with organic materials in natural waters have been studied extensively. Organo-copper interactions result in the increased solubility of some copper-containing minerals and the subsequent transport of the organocupric complex (Ong et al., 1970; Rashid and Leonard, 1973). Hydrous metal oxides can sorb copper and render it immobile (Jenne, 1968). This sorption process occurs in competition with binding of other etals, and competitive adsorption could result in the release of copper. Copper is not very mobile in sediments. Adsorption, precipitation, and organic complexation are also important processes in soils (Kabata-Pendias and Pendias, 1984). These processes render copper one of the least mobile metals.

4.1.2.6 Lead

The geochemistry of lead is dominated by its tendency to form sparingly soluble complexes with common anions such as hydroxide, sulfide, and carbonate (EPA, 1979). The divalent cation forms of lead (Pb^{+2}) are the most common in the natural environment. In unpolluted waters, lead solubility is generally controlled by the formation of lead oxide, lead sulfate and lead carbonate solids. Lead's solubility can be increased through the formation of soluble complexes with naturally-occurring organic acids. In the presence of organic acids, lead can remain complexed to fulvic acid at pH values as low as 3, whereas, in the absence of organic acids, insoluble carbonates or sulfates would be formed (Guy and Chakrabarti, 1976).

The predominant fate of lead in the environment is sorption to soils and sediments. Carey et al. (1980) found that lead concentrated in surface soils either through the formation of precipitates such as lead oxide or lead carbonate or through sorption reactions. Adsorption of lead to soils is pH dependent, increasing with increasing pH. Above pH 7, essentially all lead in

soil is sorbed (Huang et al., 1977). Korte et al. (1976) found that lead was virtually immobile in all but sandy soils. Thus, under most conditions, lead would not be expected to migrate to groundwater.

Lead can be taken up by a variety of plants, fish, and other organisms. Freshwater and marine plants and animals were observed to concentrate inorganic lead at levels 100 to 1000 times above the surrounding water (EPA, 1979). Lead uptake increases with decreasing pH, due to increased release of the element from sediments. Inorganic lead does not appear to be biomagnified in the food chain due to interactions with soil materials which render inorganic lead unavailable to biological organisms (EPA, 1979). Lead can be methylated by microorganisms in anaerobic sediments to form the compound tetramethyl lead, which is moderately volatile and mobile in the environment (Wong et al., 1975).

4.1.2.7 Manganese

Manganese (Mn) can occur in all valence states from -3 to +7. Mn(II) is very common and forms many salts. The solubility of manganese depends upon the pH of the aquatic environment. Mn(III) and Mn(IV) are only slightly soluble, though under neutral pH conditions these species may be reduced to Mn(II) which is more soluble and more mobile. Manganese can be chelated by a variety of organic and inorganic ligands. These ligands tend to keep manganese in solution. The soluble fraction of manganese ranges from 15 to 95 percent of the total, and is not dependent on pH, alkalinity, specific conductivity, or concentration of humic substances in water (Laxen et al., 1984). Soil-bound manganese can be dissolved by organic acids (Pohlman and McCall, 1986).

4.1.2.8 Mercury

Mercury (Hg) can be found in three oxidation states in the environment. Elemental mercury, Hg(0), is a liquid at ordinary temperatures. Mercurous mercury, Hg(I), occurs primarily as Hg_2^{2+} under environmental conditions and

does not form hydroxides, oxides, or sulfides. Mercuric mercury, Hg(II), forms stable complexes with common ligands. Some of these complexes are fairly soluble while others are quite insoluble (EPA, 1979). Mercuric hydroxide is not found in aquatic systems, but mercuric oxide is found and is soluble. In the presence of sulfide ion, mercuric sulfide (HgS) will precipitate from solution (Cotton and Wilkinson, 1982).

Elemental mercury is the predominant species in a moderately oxidizing environment above pH 5. Mercury becomes more soluble in the presence of chloride. Under mildly reducing conditions, HgS, which has an extremely low water solubility, will form (EPA, 1979).

The dominant process controlling mercury transport in the aquatic environment is sorption to particulates and sediments. The binding capacity of the sediment is related to its organic content; pH does not affect the sorption process. Desorption does not occur readily; therefore, mercury will tend to accumulate in sediments (Ramamoorthy and Rust, 1978).

Mercury can undergo microbial alkylation to methyl or dimethyl mercury in anaerobic or aerobic sediments. Organic forms of mercury exist in dynamic equilibrium with inorganic forms of mercury in natural waters. Methylation is of extreme importance in the environmental fate of mercury, because methylated mercury compounds are more water soluble, more easily absorbed through biological membranes, and bioaccumulated within animal tissues to a far greater extent than inorganic mercury.

Mercury has a high vapor pressure, and volatilization is a significant transport pathway for the metal. The rate of volatilization of mercury and its inorganic compounds from aquatic systems decreases in the order Hg> Hg_2Cl_2 > $HgCl_2$ > HgS > HgO. In soils, volatilization was found to increase with temperature and alkalinity (EPA, 1979).

Mercury's mobility in soils depends on the soil environment. In a study of the binding of trace metals to eleven soils, mercury was found to be

moderately to highly mobile (Korte et al., 1976). The clay content of soils does not significantly affect sorption (Kabata-Pendias and Pendias, 1984).

4.1.2.9 Selenium

The oxidation state of selenium (Se) will influence its mobility. Selenium can be found in the -2, 0, +4, and +6 oxidation states. Under oxidizing conditions over a wide pH range, selenium will be found in the +4 or +6 oxidation state as oxyanions and organo-selenium compounds. In aquatic environments, $HSeO_3^{-1}$, SeO_3^{-3}, and SeO_4^{-2} are found (EPA, 1979). Elemental selenium can also be formed over a wide pH range under mildly oxidizing to reducing conditions (Pourbaix, 1963).

In soils, pH and Eh will control selenium mobility. In acidic soils and soils with high organic matter content, the largely immobile selenides and selenium sulfides are found. In neutral, well-drained, mineral soils, selenites (Se(IV)) are found; some metal selenites, which are soluble, may also be found. In addition, selenites, as $HSeO_3^{-1}$ or SeO_3^{-2}, can be sorbed by iron oxides and removed from solution. In alkaline, well-oxidized soils, selenates (Se(VI)) will dominate. Selenates are very soluble, not well sorbed to soil particles, and hence very mobile (Kabatas-Pendias and Pendias, 1984).

Selenium sorption will be dependent upon the characteristics of the soil in which it is found. As could be expected from the above description, pH is an important variable. Frost and Griffin (1977) studied the effect of pH on selenium in landfill leachate with respect to sorption on clay materials; at high pH, they found Se(IV) was very mobile. Mobility decreased as pH decreased to pH 2 and then increased again; $HSeO_3^{-1}$ was the predominant species sorbed. Fuller (1978) reported similar results for metals sorbed as anion, which were found to be more easily mobilized than compounds sorbed as cations. Korte et al., (1976) found that the sorbing action of clays removed selenium very effectively from solution. They found selenium mobility was correlated to clay content, surface area, free ion oxide content, and pH.

4.1.2.10 Thallium

The behavior of thallium (Tl) in the environment has not been widely studied. Thallium has been found to sorb onto clays and, under reducing conditions, precipitate as the sulfide. Under aerobic conditions, thallium can form soluble salts, which increases its mobility in water (EPA, 1979).

Thallium can be found in either the +1 or +3 oxidation states. Tl(I) is more stable in water than is Tl(III). Tl(III) can form organometallic complexes, while Tl(I) forms complexes only with oxygen, sulfur, and halogen ligands (Cotton and Wilkinson, 1982). Tl(I) is the dominant oxidation state under mildly oxidizing to reduced conditions. Under very reducing conditions, thallium may be precipitated as the metal, or in the presence of sulfur, as the sulfide (EPA, 1979).

Montmorillonite clays have been found to sorb thallium (Magorian et al., 1974). Adsorption was more effective at pH 8.1 than at pH 4. Under basic conditions, thallium was found to sorb onto ferric oxide. Either of these types of interactions is likely to occur in soils. Because thallium is not a well-studied element, it is difficult to determine the behavior of thallium in the environment. Thallium is likely to be mobile in solution, but adsorption to soil particles may also occur.

Thallium's metallic and covalent radii are similar to those of lead. In addition, the univalent ionic radius of thallium is similar to those of potassium and rubidium. Therefore, it is likely to thallium will behave like lead, potassium, or rubidium depending on pH, Eh, and the presence of other ligands.

4.2 MECHANISMS OF MIGRATION

There are a number of mechanisms by which chemicals of potential concern may migrate from contaminated areas of WDI to on-site or off-site receptors. Migration of chemicals to air may occur via fugitive dust

emissions or volatilization. Migration of chemicals to groundwater could occur via vertical and horizontal routes. Subsurface soil contaminants may leach downward to groundwater as a result of rain water infiltration; contaminants in shallow aquifers may migrate through areas of hydraulic continuity to deeper aquifers. Horizontal migration of groundwater contaminants may occur as the result of groundwater flow. Chemical vapors and landfill gases could also migrate through permeable zones or utility lines. The potential for the chemicals of potential concern to migrate via these mechanisms is described in the following subsections.

4.2.1 MIGRATION INTO AIR

Fugitive dust emissions of contaminated particulates could occur in areas of the site that are not completely paved or covered with vegetation, such as the reservoir, the waste handling areas, and Toxo Spray Dust. Particulate emissions would be of potential concern primarily for inorganic chemicals, and for those organic chemicals which are relatively nonvolatile, such as PAHs, pesticides, and phthalates.

Organic chemicals, such as vinyl chloride, have been detected in subsurface gas in the reservoir. At this time, the integrity of the reservoir is unknown; potential exists for the release of these volatile contaminants through cracks in the reservoir as well as via upward migration. Permeable soils in the area along with local features of the subsurface, such as permeable zones (buried utility lines, sewers) or areas of impermeable soil cover (paving, buildings) may provide conduits for subsurface gas migration. These gases may ultimately discharge to the soil surface and become mixed with the ambient air, or may seep into buildings along the foundation. The direction of movement and degree of dilution before reaching a receptor have not been determined, so contaminants will be assumed to disperse radially over the WDI site.

4.2.2 PERCOLATION INTO GROUNDWATER

The groundwater transport potential of each of the classes of chemicals of potential concern at the WDI site was discussed in Section 4.1. Several aquifers underlie the WDI site. Contamination has been detected in the uppermost aquifer. One deep well was installed at the WDI site during the RI. Additional wells were not installed since contamination was not suspected (EBASCO, 1989c). Chemicals may potentially migrate vertically from the shallow aquifer to lower aquifers by both manmade (e.g., abandoned oil wells) and natural (e.g., hydraulic continuity) conduits, and 2) horizontally by the flow of the aquifer. Contaminants in subsurface soils can also leach from soils and migrate vertically into groundwater. The extent to which these chemicals will actually migrate depend upon a number of site-specific factors, including the direction and rate of groundwater flow, the physical and chemical makeup of the saturated and unsaturated soils beneath the site, and the geochemistry and microbiology of the subsurface environment. Many of the organic chemicals of potential concern are susceptible to rapid chemical or biological transformation in soils, and therefore may not reach groundwater. The solubilities of the inorganic chemicals of potential concern may vary depending on the pH and redox conditions prevailing in the area.

4.3 POTENTIAL HUMAN EXPOSURE PATHWAYS

In order for a chemical to pose a human health risk, a complete exposure pathway must be identified. A complete exposure pathway consists of four elements: 1) a source and mechanism of chemical release to the environment, 2) an environmental transport medium (e.g., air, soil) for the released chemical, 3) a point of potential human contact with the contaminated medium (known as the exposure point), and 4) a human exposure route (e.g., inhalation) at the contact point (EPA, 1986a). This exposure analysis will be developed from two different perspectives. The first will consider current land use in the area and the second will consider potential future uses that may differ from the current situation. As discussed in Section 2.2, the WDI site lies adjacent to the St. Paul High School athletic field and local

businesses; residences are located immediately south of the site. Individuals living, working, or attending school in the vicinity of the WDI site may be potentially exposed to site contaminants through a number of exposure pathways. Worker exposures will not be evaluated in this assessment since these exposures are outside the scope of CERCLA/SARA.

As discussed above, individuals may be exposed to contaminants associated with the WDI site through a variety of pathways. In order to quantify human health risks, it is necessary to estimate the concentration of the contaminant at the point of human contact. This concentration is known as the exposure point concentration. For those media where the exposure point is at the sample collection point (e.g., surface soil), the geometric mean and maximum measured concentrations presented in Section 2.4 will be used as the exposure point. For those exposure scenarios where the exposure point is not the sampled media, contaminant migration modeling is required to estimate the exposure point concentrations. The methods of estimating the exposure point concentrations are discussed below.

In this section, exposure pathways and exposure point concentrations are discussed for each of the study areas of potential concern at the WDI site. Potential exposure pathways are evaluated for each potentially contaminated environmental medium: soil, groundwater, surface water, and air.

4.3.1 CURRENT-USE CONDITIONS

As discussed previously, the WDI property is vacant and no longer operates as a disposal site. However, schools, businesses, and residences are located along the perimeter of the former disposal site. Students and residents may be exposed to contaminants migrating from the site. Although the area surrounding the former reservoir is fenced, school children are known to have trespassed on the property. School children may come in contact with site contaminants while trespassing. Off-site residents may be exposed to airborne particulates generated by wind erosion. Discussed below are potential current-use exposure pathways for contaminants in soil, groundwater,

surface water, and air. Table 4-5 evaluates the current-use exposure pathways.

4.3.1.1 Surface Soil Pathways

As discussed in Section 2.4.2.1, chemical contaminants have been detected in surface soils throughout the site. Potential routes of exposure to contaminated surface soils include direct soil contact and inhalation of contaminated airborne particulates. The direct contact pathway will be discussed in this section for current conditions. Inhalation of airborne particulates will be discussed under the air pathways in Section 4.3.1.4.

Under current-use conditions, individuals trespassing on the WDI site may be exposed to soil-borne contaminants via direct contact with contaminated soils. Direct contact with contaminated soil can lead to exposure either via inadvertent or intentional soil ingestion or via dermal absorption. The direct ingestion of contaminated soil is especially of concern for young children, who may ingest contaminated dirt by normal mouthing of soiled objects or their fingers and/or hands (Mahaffey, 1978; DHS, 1987). Older children are less likely to eat soil or to mouth soiled objects, but they still may ingest dirt from their hands. Adults on site may ingest some contaminated soil, but they are less likely to be exposed by this route, unless they have a high incidence of hand-to-mouth contact (e.g., smokers, gardeners). School-aged children may contact contaminated surface soils of the WDI reservoir and sump areas while trespassing. The Toxo Spray Dust site is fenced and locked; therefore, trespassing in this area is considered unlikely.

For those scenarios involving exposure to surface soils, measured surface soil concentrations were used to predict exposure. For some chemicals (e.g., PAHs, PCBs, and some chlorinated pesticides), toxicity values (e.g., CPFs, RfDs) were not available for each chemical constituent or isomer. In these cases, geometric mean concentrations of the chemicals in each class were summed to represent the overall average concentration for the class; the sum

TABLE 4-5
HUMAN EXPOSURE PATHWAYS FOR WDI
UNDER CURRENT USE CONDITIONS

Environmental Media	Exposure Point	Potential Receptors	Route of Exposure	Pathway Complete?
Surface Soil	WDI Site	Trespassers	Direct Contact	Yes. School children have trespassed on the WDI property.
Groundwater				No. There are no identified users of the contaminated aquifers under current-use conditions.
Surface Water				No. There are no identified surface waters on or near the WDI site.
Air	WDI Site	Off-Site Residents	Subsurface Gas Migration	Yes. Contaminants may migrate upward to the ground surface and be released to ambient air.
	WDI Site		Airborne Particulates	Yes. Wind dispersion of contaminated unpaved surface soils may result in airborne particulates.
	St. Paul's High School	Students	Subsurface Gas Migration	Yes. Contaminants may migrate upward to the ground surface and be released to ambient air.
	St. Paul's High School		Airborne Particulates	Yes. Wind dispersion of contaminated unpaved surface soils may result in airborne particulates.
		Trespassers	Airborne Particulates	No. Unless winds are high during trespassing incidents, this pathway is anticipated to be insignificant in relation to direct contact.

of the maximum concentrations was used for the plausible maximum case. A representative chemical with an available toxicity value was then selected. The PAHs were segregated into two groups, carcinogenic and noncarcinogenic based on the discussion in Section 3.2. The carcinogenic PAHs were summed with benzo(a)pyrene selected as the representative for that group. Benzo(a)pyrene is considered to be one of the most potent of the carcinogenic PAHs and therefore, assuming that all the carcinogenic PAHs are benzo(a)pyrene will generate health-conservative risk estimates. Noncarcinogenic PAHs will be represented by naphthalene. Concentrations of DDD, DDE, and DDT will be summed and represented by DDT. For PCBs, most of the toxicity data are based on Aroclor mixtures, rather than an individual isomer; it is therefore, more realistic to evaluate PCBs as a group. The concentrations of the alpha- and gamma isomers of chlordane were summed to evaluate overall chlordane exposure.

Concentrations of soil contaminants to be used in this assessment are presented in Table 4-6.

4.3.1.2 Groundwater Pathways

Volatile organic chemicals and inorganics have been detected in the aquifer underlying WDI as presented in Section 2.4.2.2. At this time, there are no identified users of this aquifer; however, as discussed in Section 2.3.4, this aquifer may potentially be hydraulically interconnected to aquifers which provide water for domestic uses. Exposure to users of water from the shallow aquifer will be considered under future-use conditions.

4.3.1.3 Surface Water Pathways

No surface waters have been identified on or near the WDI site. Therefore there are no potential exposure pathways for surface water.

TABLE 4-6
SOIL EXPOSURE POINT CONCENTRATIONS USED TO DETERMINE
RISKS ASSOCIATED WITH THE CURRENT USE
DIRECT CONTACT PATHWAYS FOR TRESPASSERS
WDI SITE

Chemical	CONCENTRATION	
	Average	Maximum
CARCINOGENS		
INORGANICS	(mg/kg)	(mg/kg)
Arsenic	8.0	85
ORGANICS	(ug/kg)	(ug/kg)
Benzene	260	260
Chlordane	10	210
DDT/DDE/DDD	140	3,940
Dieldrin	38	280
Heptachlor Epoxide	9.6	46
Methylene Chloride	23	1400
PAHs - Carcinogenic	890	2,420
PCBs	2,100	4,900
Pentachlorophenol	250	290
NONCARCINOGENS		
INORGANICS	(mg/kg)	(mg/kg)
Antimony	5.4	7.6
Arsenic	8	85
Cadmium	1.1	3.3
Chromium	23	53
Copper	36	511
Lead	43	731
Manganese	364	581
Mercury	0.15	1.2
Selenium	0.50	1.2
Thallium	7.9	19
ORGANICS	(ug/kg)	(ug/kg)
Benzoic Acid	150	230
2-Butanone	8.2	46
Chlordane	10	210
DDT	140	3940
Ethylbenzene	47	1,500
Heptachlor Epoxide	9.6	46
Methylene Chloride	23	1,400
PAHs- Noncarcinogenic	4,420	17,910
Pentachlorophenol	250	290
Toluene	52	9,000
Xylene	190	2,500

4.3.1.4 Air Pathways

Contaminants may be released from the WDI site to ambient air in two ways: via the migration of subsurface gas to the ground surface and subsequent dispersion in ambient air or the wind entrainment of airborne particulates from surface soils. Subsurface volatile contaminants may be released to the air through unpaved areas resulting in exposure to students, workers, and residents. Vinyl chloride and other volatile chemicals have been detected in subsurface gases at the site. Most of the WDI site is unpaved; therefore, particulate-bound contaminants could be suspended in the air by wind erosion or traffic on the site. Metals, PAHs, and pesticides are most likely to be bound to soils.

In order for individuals to be exposed to subsurface gas contaminants, these contaminants must migrate upward toward the ground surface and be released into ambient air.

Subsurface gas samples were collected from subsurface gas wells screened between 5 and 35 feet below ground surface. Since it is unlikely that individuals will be exposed directly to subsurface gas contaminants, models will be used to estimate exposure point concentrations for current and future-use conditions. In both current and future-use scenarios, the average cases will use geometric means calculated using all samples; the plausible maximum cases will use the geometric mean of positively detected samples.

Once emitted to ambient air, contaminants must be transported to a downwind receptor. In order to estimate exposure point concentrations under current use conditions, two models were employed. The first, a variation of an equation developed by Farmer et al. (1978; EPA, 1988e), was used to predict the concentration released at the ground surface. A virtual point source Gaussian dispersion model (Turner, 1970) was used to estimate exposure point concentrations to receptors at distances of 0.1, 0.5, and 1 km downwind of the WDI site. Emissions were based on geometric mean concentrations since mixing is assumed to reduce the maximum concentrations prior to contact with the

receptor. Methods used to estimate current exposure point concentrations are described in Appendix B. Current use scenario exposure point concentrations for subsurface gas contaminants are presented in Table 4-7.

Under current-use conditions, individuals living or attending school off site may be exposed to WDI contaminants as a result of inhalation of particulates generated from contaminated surface soils. Wind erosion of particulates was estimated using a procedure developed by Cowherd et al. (1984). Soil concentrations presented in Table 4-6 were used to estimate particulate emissions. Details of the procedure used are presented in Appendix B. Once particulates are emitted, a virtual point source Gaussian dispersion model (Turner, 1970) was used to estimate exposure point concentrations for receptors at distances of 0.1, 0.5, and 1 km from the site. Exposure point concentrations for airborne particulates are listed in Table 4-8. In this assessment, all chemicals in surface soils will be evaluated as particulate contaminants.

4.3.2 FUTURE-USE CONDITIONS

Under future-use conditions, the WDI site may be developed as commercial or residential property. If this were to occur without remediation of the site, on-site workers and residents may potentially be exposure to site contaminants. In this section, potential future uses of the WDI site are discussed and the resulting exposure pathways evaluated for soil, groundwater, and air. Table 4-9 evaluates potential future exposure pathways.

4.3.2.1 Soil Pathways

If the WDI site were developed for commercial or residential purposes, on-site workers and residents may come in direct contact with contaminated surface soils. Workers involved in construction at the site may have additional short-term exposure to both surface and subsurface contaminants during excavating activities. Evaluation of exposure during site remediation is outside the scope of this assessment and will not be considered further.

TABLE 4-7

CONCENTRATIONS OF AIRBORNE SUBSURFACE GAS ESTIMATED FOR DETERMINING RISKS ASSOCIATED WITH CURRENT INHALATION FOR RESIDENTS AND STUDENTS

Chemical	(a) Average Case (mg/m3) at 0.1 km	at 0.5 km	at 1 km	(b) Plausible Maximum Case (mg/m3) at 0.1 km	at 0.5 km	at 1 km
CARCINOGENS						
Benzene	6.0E-06	1.1E-06	5.9E-07	5.1E-05	9.6E-06	5.0E-06
Carbon Tetrachloride	9.5E-08	1.8E-08	9.3E-09	8.4E-07	1.6E-07	8.3E-08
Chloroform	2.4E-07	4.4E-08	2.3E-08	4.5E-06	8.4E-07	4.4E-07
1,2-Dibromoethane	9.6E-06	1.8E-06	9.4E-07	2.0E-05	3.8E-06	2.0E-06
1,2-Dichloroethane	2.8E-06	5.2E-07	2.7E-07	1.2E-05	2.3E-06	1.2E-06
Tetrachloroethylene	4.6E-06	8.5E-07	4.4E-07	7.3E-06	1.4E-06	7.1E-07
Trichloroethylene	8.5E-06	1.6E-06	8.3E-07	2.0E-05	3.7E-06	1.9E-06
Vinyl Chloride	2.8E-06	5.3E-07	2.8E-07	1.1E-04	2.1E-05	1.1E-05
NONCARCINOGENS						
1,1,1-Trichloroethane	2.4E-06	4.6E-07	2.4E-07	1.2E-05	2.2E-06	1.1E-06

(a) - Based on geometric mean of all samples.

(b) - Based on geometric mean of positively detected samples.

TABLE 4-8

EXPOSURE POINT CONCENTRATIONS OF AIRBORNE PARTICULATES ESTIMATED FOR DETERMINING RISKS ASSOCIATED WITH CURRENT INHALATION PATHWAY FOR BOTH RESIDENTS AND STUDENTS

WDI SITE

Chemical	ESTIMATED AMBIENT AIR CONCENTRATION (mg/m3) at 0.1 km	at 0.5 km	at 1.0 km
CARCINOGENS			
INORGANICS			
Arsenic	6.0E-07	1.1E-07	5.8E-08
Cadmium	8.1E-08	1.5E-08	7.9E-09
Chromium	1.7E-06	3.2E-07	1.7E-07
ORGANICS			
Benzene	1.9E-08	3.6E-09	1.9E-09
Chlordane	7.4E-10	1.4E-10	7.3E-11
DDT	1.0E-08	1.9E-09	1.0E-09
Dieldrin	2.8E-09	5.3E-10	2.8E-10
Heptachlor Epoxide	7.1E-10	1.3E-10	7.0E-11
Methylene Chloride	1.7E-09	3.2E-10	1.7E-10
PAHs	6.6E-08	1.2E-08	6.5E-09
PCBs	1.5E-07	2.9E-08	1.5E-08
Pentachlorophenol	1.8E-08	3.4E-09	1.8E-09
NONCARCINOGENS			
INORGANICS			
Antimony	4.0E-07	7.5E-08	3.9E-08
Copper	2.7E-06	5.0E-07	2.6E-07
Lead	3.2E-06	6.0E-07	3.1E-07
Mercury	1.1E-08	2.1E-09	1.1E-09
Selenium	3.7E-08	7.0E-09	3.6E-09
Thallium	5.9E-07	1.1E-07	5.8E-08
ORGANICS			
2-Butanone	6.1E-10	1.1E-10	6.0E-10
Ethylbenzene	3.5E-09	6.6E-10	3.4E-10
PAHs - Noncarcinogenic	3.3E-07	6.2E-08	3.2E-08
Pentachlorophenol	1.8E-08	3.4E-09	1.8E-09
Toluene	3.9E-09	7.3E-10	3.8E-10
Xylenes	1.4E-08	2.7E-09	1.4E-09

TABLE 4-9
HUMAN EXPOSURE PATHWAYS FOR WDI
UNDER FUTURE USE CONDITIONS
WDI SITE

Environmental Media	Exposure Point	Potential Receptors	Route of Exposure	Pathway Complete?
Surface Soil	WDI Site	Residents	Direct Contact	Yes. Residents would be exposed to surface soil contaminants while gardening or playing.
Groundwater	WDI Site	Residents	Ingestion	Yes. Water from this area could be blended with other water sources and result in exposure to contaminants. Also, contaminants may migrate from the upper aquifers to lower aquifers through existing conduits.
Surface Water				No. There are no identified surface waters on or near the WDI site.
Air	WDI Site	On-site Residents	Subsurface Gas	Yes. If houses are constructed on land overlying the reservoir.
			Airborne Particulates	No. If the property were developed, pavement and vegetation would prevent the generation of airborne particulates.
	St. Paul's High School	Students	Subsurface Gas	Yes. Same as current use conditions.

Since residents are expected to be present at the site for longer time periods, they are anticipated to have the greatest potential exposure, and, therefore they will be the receptor group evaluated in this assessment. Locations on the WDI site are known to be 10^{-2} x 0 feet above grade due to the presence of fill material. In evaluating exposures to future on-site residents, it will be assumed that residents may potentially be exposed to contaminants present in the first 20 feet of soil as a result of grading. Concentrations used to evaluate the soil pathway are listed in Table 4-10.

Under future-use conditions, individuals are unlikely to contact the deeper contaminated soils, except during site remediation. Contaminants in subsurface soils may migrate into groundwater as a result of rainwater infiltration. Sampling results from the groundwater investigation did not reveal the presence of soil contaminants in groundwater underlying the site. This may indicate that contaminants in subsurface soils are relatively immobile and the reservoir has been an effective barrier to contaminant migration or that groundwater contaminants have already migrated off site. However, due to the large volume of soil contamination, it is unlikely that the highest concentrations of contaminants would be found off site. Another route of soil contaminant transport is via subsurface gas migration. This route will be discussed in Section 4.3.2.3.

4.3.2.2 Groundwater Pathways

In the future, on-site residents may install a domestic well in the shallow aquifer underlying the WDI site or contaminants in the upper aquifer may migrate vertically into groundwater used for domestic water supply. If this occurs, users of this water may potentially be exposed to contaminants emanating from the WDI site. The installation of domestic water wells in the upper aquifer by on-site residents will be quantified in this assessment. Groundwater concentrations reported in Table 4-11 will be used in this assessment.

TABLE 4-10
SOIL EXPOSURE POINT CONCENTRATIONS USED TO DETERMINE
RISKS ASSOCIATED WITH THE FUTURE USE DIRECT CONTACT PATHWAYS
WDI SITE

Chemical	CONCENTRATION	
	Average	Maximum
CARCINOGENS		
INORGANICS	(mg/kg)	(mg/kg)
Arsenic	6.5	337
ORGANICS	(ug/kg)	(ug/kg)
Aldrin	23	23
Benzene	110	19,000
BHC (delta & gamma isomers)*	485	995
Carbon Tetrachloride	2	2
Chlordane (alpha & gamma isomers)	79	2,060
Chloroform	1.6	5
DDT/DDE/DDD	206	352,000
Dieldrin	63	280
1,4-Dichlorobenzene	170	2,400
Heptachlor	87	87
Heptachlor Epoxide	9.6	46
Methylene Chloride	18	1,200
PAHs - Carcinogenic	1,340	13,460
PCBs	1,130	5,550
Pentachlorophenol	240	320
Tetrachloroethylene	28	43,000
Trichloroethylene	140	5,000
Vinyl Chloride	31	1,700

TABLE 4-10 (cont'd)
SOIL EXPOSURE POINT CONCENTRATIONS USED TO DETERMINE
RISKS ASSOCIATED WITH THE FUTURE USE DIRECT CONTACT PATHWAYS
WDI SITE

Chemical	CONCENTRATION	
	Average	Maximum
NONCARCINOGENS		
INORGANICS	(mg/kg)	(mg/kg)
Antimony	5.2	25
Arsenic	6.5	337
Cadmium	1.0	18
Chromium	26	149
Copper	30	721
Lead	20	2,790
Manganese	400	2,270
Mercury	0.13	11
Selenium	0.41	1
Thallium	14	39
Zinc	83	490,490
ORGANICS	(ug/kg)	(ug/kg)
Aldrin	23	23
gamma-BHC *	15	15
Benzoic Acid	170	1,300
2-Butanone	16	11,000
Carbon Tetrachloride	2	2
Chlordane	79	2,060
Chloroform	1.6	5
DDT/DDE/DDD	206	352,000
1,4-Dichlorobenzene	170	2,400
Dieldrin	63	280
Ethylbenzene	160	30,000
Heptachlor	87	87
Heptachlor Epoxide	9.6	46
Methylene Chloride	18	1,200
PAHs- Noncarcinogenic	3,169	302,930
Pentachlorophenol	240	320
Tetrachloroethylene	28	43,000
Toluene	77	120,000
1,1,1-Trichloroethane	500	1,800
Trichloroethylene	140	5,000
Xylenes	380	250,000

* Only the gamma isomer of BHC has a noncarcinogenic toxicity value.

TABLE 4-11.
EXPOSURE POINT CONCENTRATIONS OF CHEMICALS
OF POTENTIAL CONCERN IN GROUNDWATER

WDI SITE

A. POTENTIALLY CARCINOGENIC CHEMICALS

Chemical	Concentration (ug/liter) Geometric Mean	Maximum
INORGANICS		
Arsenic	5.9	12
ORGANICS		
Chloroform	2.8	9
Tetrachloroethylene	2.6	11
Trichloroethylene	2.7	18

B. NONCARCINOGENIC CHEMICALS

Chemical	Concentration (ug/liter) Geometric Mean	Maximum
INORGANICS		
Arsenic	5.9	12
Lead	4.0	16
Manganese	495	5850
Mercury	0.15	2.0
ORGANICS		
Chloroform	2.8	9
Tetrachloroethylene	2.6	11
Toluene	2.6	5.0
Trichloroethylene	2.7	18

4.3.2.3 Air Pathways

The contaminant release mechanisms discussed under current-use will be applicable under future-use conditions as well. However, if on-site residences are established, the potential for wind erosion of particulates is greatly reduced due to the presence of buildings and vegetation on-site. Therefore, under future-use conditions, particulate exposure will not be quantified. Under future-use conditions, on-site residents may potentially be exposed to subsurface gas contaminants which have infiltrated through the foundation into the house. The potentially exposed population will increase if the WDI property is developed for commercial or residential purposes. This residential population may also include sensitive individuals such as asthmatics, young children and the elderly which may be more adversely affected by airborne contaminants.

Contaminants present in subsurface gas may be the result of volatilization of contaminants from subsurface soils and groundwater. Since extensive soil contamination and limited groundwater contamination was detected, it will be assumed that soils are the primary subsurface gas source. In this assessment, concentrations of contaminants in subsurface gas will be used to evaluate exposure to volatile contaminants in subsurface soils. Since the subsurface gas wells are screened from 5 to 35 feet, these data are assumed to be representative of the volatilization occurring in the subsurface.

Under future-use conditions, subsurface gas contaminants may infiltrate into on-site residences resulting in exposure through inhalation of indoor air. In this assessment, the geometric mean of measured subsurface gas concentrations was assumed to occur at the foundation of the house. Subsurface gas is assumed to infiltrate into houses at a rate of 1 m^3/hr (Scott, 1983). Once the emission rates are determined, the associated indoor air concentration was estimated by using a one-compartment indoor air model. These models are detailed in Appendix B. Exposure point concentrations for the indoor air scenario are presented in Table 4-12.

TABLE 4-12
EXPOSURE POINT CONCENTRATIONS OF SUBSURFACE GAS CONTAMINANTS
FOR ON-SITE RESIDENTS
WDI SITE

COMPOUND	Average Case Indoor Air Concentration (a) (ug/m3)	Plausible Maximum Case Indoor Air Concentration (b) (ug/m3)
Benzene	1.2E+00	6.3E+00
Carbon Tetrachloride	2.1E-02	1.1E-01
Chloroform	4.6E-02	5.4E-01
1,2-Dibromoethane	1.9E+00	2.5E+00
1,2-Dichloroethane	5.1E-01	1.4E+00
Tetrachloroethene	1.1E+00	1.1E+00
1,1,1-Trichloroethane	5.3E-01	1.6E+00
Trichloroethene	1.8E+00	2.6E+00
Vinyl Chloride	4.6E-01	1.1E+01

(a) Based on geometric mean of all samples.
(b) Based on geometric mean of positively detected samples only.

5.0 RISK CHARACTERIZATION

According to guidelines for preparing risk assessments as part of the RI/FS process (EPA, 1986a; DHS, 1986), the potential adverse effects on human health should be assessed, where possible, by comparing chemical concentrations found at or near the site with applicable or relevant and appropriate requirements (ARARs) or other guidance that have been developed for the protection of human health or the environment. If ARARs are available for all chemicals in all environmental media, then a comparison to ARARs constitutes the risk assessment. If not, quantitative risk estimates must be developed in addition to the comparison to ARARs. ARARs or other guidance are available only for a few of the site-related chemicals in groundwater and air at WDI; therefore, quantitative risk characterization will be completed for human exposure to soil, air, and groundwater in this section. A comparison with potential ARARs and other guidance and a discussion of uncertainties and their effects on risk are presented below. A qualitative ecological assessment is also presented in this section.

5.1 APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARs)

Remedial actions selected under the Superfund Amendments and Reauthorization Act of 1986 (SARA) must attain levels of cleanup of hazardous substances released into the environment and of control of further release which assure protection of human health and environment. SARA specifies that any selected remedial action must achieve a level of control which at least attains requirements that are legally applicable to the hazardous substances of concern or relevant and appropriate under the circumstances of release or threatened release. Accordingly, EPA guidelines for preparing risk assessments as part of the RI/FS process (EPA, 1986a) recommend comparison of chemical concentrations found at or near a site with ARARs. Therefore, EPA's interim guidance on ARARs (EPA, 1987i) will be followed for this assessment; ARARs are defined as follows:

- Applicable Requirements means those cleanup standards, standards of control, and other substantive environmental protection

requirements, criteria, or limitations promulgated under Federal or State law that specifically address a hazardous substance, pollutant, contaminant, remedial action, location, or other circumstance at a CERCLA site. "Applicability" implies that the remedial action or the circumstances at the site satisfy all of the jurisdictional prerequisites of a requirement....

- Relevant and appropriate requirements means those cleanup standards, standards of control, and other substantive environmental protection requirements, criteria, or limitations promulgated under Federal or State law that, while not "applicable" to a hazardous substance, pollutant, contaminant, remedial action, location, or other circumstance at a CERCLA site, address problems or situations sufficiently similar to those encountered at the CERCLA site that their use is well suited to the particular site.

  The relevance and appropriateness of a requirement can be judged by comparing a number of factors, including the characteristics of the remedial action, the hazardous substances in question, or the physical circumstances of the site, with those addressed in the requirement. It is also helpful to look at the objective and origin of the requirement. For example, while RCRA regulations are not applicable to closing undisturbed hazardous waste in place, the RCRA regulation for closure by capping may be deemed relevant and appropriate.

  A requirement that is judged to be relevant and appropriate must be complied with to the same degree as if it were applicable. However, there is more discretion in this determination: it is possible for only part of a requirement to be considered relevant and appropriate, the rest being dismissed if judged not to be relevant and appropriate in a given case.

  Non-promulgated advisories or guidance documents issued by Federal or State governments do not have the status of potential ARARs. However, ..., they may be considered in determining the necessary level of cleanup for protection of health or environment.

Only those ARARs or advisories or guidance that are ambient or chemical-specific requirements (i.e., those requirements which "set health or risk based concentration limits or ranges in various environmental media for specific hazardous substances, pollutants, or contaminants" (EPA, 1987i)) as opposed to ARARs which are classified as action-specific or locational, are used in risk assessments. Under SARA, EPA at a minimum currently considers maximum contaminant levels (MCLs) developed under the Safe Drinking Water Act, National Ambient Air Quality Standards (NAAQS), and State drinking water and

air standards to be potential ARARs for use in risk assessment at CERCLA/SARA sites.

In addition, other relevant criteria or guidance values, such as EPA's Ambient Water Quality Criteria (AWQCs) developed under the Clean Water Act and the maximum contaminant level goals (MCLGs) established under the Safe Drinking Water Act, may be useful in assessing baseline risks or developing goals for remedial action. The California Department of Health Services (DHS) has also developed media-specific advisory levels, known as Applied Action Levels (AALs) for use in the California Site Mitigation Decision Tree (DHS, 1986). The AALs for air and water will also be considered as guidance values in this EA. Potential chemical-specific ARARs for the WDI site are identified below.

5.1.1 POTENTIAL ARARS AND OTHER GUIDANCE

In this section, potential ARARs and other guidance values are identified for all complete exposure pathways (soil, groundwater, and air) under current and future use conditions. The contaminant specific ARARs are then compared to measured and/or modeled exposure point contaminant concentrations.

5.1.1.1 Soil

Currently, there are no health related ARARs (e.g., statutory requirements) regarding soil contamination levels for direct contact with soil contaminants. Guidance values are available for lead in soil. EPA (1989f) has set an interim soil cleanup level for total lead at 500 mg/kg to 1000 mg/kg, which is considered to be protective for direct contact at residential settings. The guidance is based on a recommendation by the centers for Disease Control (CDC) which states that "...lead in soil and dust appears to be responsible for blood levels in children increasing above background levels when the concentration in the soil or dust exceeds 500 to 1000 ppm" (ATSDR, 1988b). Lead concentrations detected in WDI surface soils ranged from a

geometric mean of 41 mg/kg to a maximum of 731 mg/kg. The maximum concentration exceeds the 500 mg/kg advisory value, though the average over all site surface soils is less than the advisory level by a factor of ten. The California Department of Health Services is in the process of developing Applied Action Levels (AALs) for soil contaminants; however at this time, no soil AALs are available.

In its definition of hazardous wastes, the State of California has established chemical-specific Total Threshold Limit Concentrations (TTLCs) for many hazardous materials. The TTLCs for chemicals found in soils at the WDI site are listed in Table 5-1. The maximum concentration of lead, zinc, and DDT in soils 0 to 20 feet exceeded their respective TTLCs. The maximum concentration of DDT in surface soil also exceeded the TTLC. No other chemicals exceeded their TTLC values.

5.1.1.2 Groundwater

For groundwater which is currently used or may be used in the future as a municipal drinking water supply, Federal and State MCLs are applicable as ARARs. EPA (1986f) established a Groundwater Protection Strategy which classifies groundwater based on its use, value to society and vulnerability to contamination. Class I groundwater is defined as highly vulnerable and either an irreplaceable source of drinking water (e.g., federally designated sole source aquifer) or ecologically vital. Class II groundwater is all non-Class I groundwater that is currently used or potentially available for drinking water or other beneficial use. Class IIA designates current drinking water sources; Class IIB identifies potential drinking water sources. Class III is not a potential source of drinking water and of limited beneficial use. MCLs are, therefore, applicable to Class I and II aquifers.

The uppermost aquifer underlying the WDI site is best classified as Class IIB groundwater. Groundwater from this aquifer has been used by private well owners in the past for drinking water. Groundwater from the deeper aquifers is currently used as municipal drinking water; since this water is

TABLE 5-1
TOTAL THRESHOLD LIMIT CONCENTRATIONS (TTLC) FOR SOIL CONTAMINANTS
WDI SITE

Chemical	TTLC [a] (mg/kg)	ON-SITE CONCENTRATIONS			
		Surface Soils (mg/kg)		Soils(0-20 ft) (mg/kg)	
		Average	Maximum	Average	Maximum
INORGANICS					
Antimony	500	5.4	7.6	5.2	25
Arsenic	500	8.0	85.1	6.5	337
Cadmium	100	1.1	3.3	1.0	18.2
Chromium	500 [b]	23	53	26	149
Copper	2,500	36	511	30	721
Lead (inorganic)	1,000	43	731	20	2,790
Manganese	NS [c]	364	581	400	2,270
Mercury	20	0.15	1.2	0.13	11
Selenium	100	0.50	1.2	0.41	1.2
Thallium	700	7.9	18.7	14.0	39
Zinc	5,000	101	304	83	490,490
ORGANICS					
Aldrin	1.4	ND [d]	ND	0.023	0.023
Benzene	NS	0.26	0.26	0.11	19
BHC (gamma & delta isomers)	4.0	ND	ND	0.485	0.995
Benzoic Acid	NS	0.15	0.23	0.17	1.3
2-Butanone	NS	0.0082	0.046	0.016	11
Chlordane	2.5	0.01	0.21	0.079	2.06
DDT	1.0	0.14	3.94	0.206	352
1,4-Dichlorobenzene	NS	ND	ND	0.17	2.4
Dieldrin	8.0	0.038	0.28	0.063	0.28
Ethylbenzene	NS	0.047	1.5	0.16	30
Heptachlor	4.7	ND	ND	0.087	0.087
Heptachlor Epoxide	NS	0.0096	0.046	0.0096	0.046
Methylene Chloride	NS	0.023	1.4	0.018	1.2
PAHs - Carcinogenic	NS	0.89	2.42	1.34	13.46
PAHs - Noncarcinogenic	NS	4.22	18.11	3.169	302.93
PCBs	50	2.1	4.9	1.13	5.55
Pentachlorophenol	17	0.25	0.29	0.24	0.32
Toluene	NS	0.052	9	0.077	120
Xylenes	NS	0.19	2.5	0.38	250

[a] California Administrative Code (CAC). Title 26. Section 22-66699.
[b] Chromium (VI) compounds.
[c] NS = No Standard.
[d] ND = Not Detected.

blended with water from other sources, these aquifers are most appropriately classified Class IIA.

Federal maximum contaminant levels (MCLs) promulgated under the Safe Drinking Water Act and California drinking water standards are ARARs for the aquifers underlying the WDI site and are presented in Table 5-2. Of all the inorganic chemicals of concern in groundwater, only two were detected at levels significantly above their ARARs. Lead was detected at a maximum concentration which exceeded the California MCL and proposed Federal MCL of 5 μg/liter by approximately 3 times; the upgradient geometric mean concentration of lead (6.3 μg/liter) also exceeded these values. Geometric mean and maximum concentrations of manganese exceeded the California secondary MCL by approximately 10 to 200 times, respectively. THe upgradient concentration of manganese (211 μg/liter) exceeded the secondary MCL as well. It should be noted that secondary standards are based on consumer acceptability (i.e., taste and odor) considerations rather than on protection of human health. Arsenic and mercury did not exceed their ARARs.

Of the organic compounds with available ARARs, only the concentrations of tetrachloroethylene and trichloroethylene exceeded their ARARs. In both cases, the maximum detected concentration exceeded the California MCL; tetrachloroethylene also exceeded the proposed Federal MCL by approximately two times. Neither chloroform nor toluene exceeded their ARARs.

Other potential criteria and guidance values for these aquifers include maximum contaminant level goals (MCLGs) developed under the Safe Drinking Water Act, Ambient Water Quality Criteria (AWQC) developed under the Clean Water Act and California AALs for water. These are presented in Table 5-3.

5.1.1.3 Air

ARARs for air contaminants include national ambient air quality standards (NAAQS) promulgated under the Clean Air Act and California ambient air quality standards (CAAQS) promulgated under California's Clean Air Act.

TABLE 5-2
APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARS)
FOR CHEMICALS OF CONCERN IN GROUNDWATER
AT THE WASTE DISPOSAL, INC. SITE

Chemical	Federal MCL (µg/L)	Federal Secondary MCL (µg/liter)	Proposed Federal MCL[a] (µg/liter)	Proposed Secondary Federal MCL[a] (µg/liter)	California MCL (µg/liter)	California Secondary MCL (µg/liter)
INORGANICS						
Arsenic	50	--	--	--	50	--
Lead	50	--	5[b]	--	5	--
Manganese	--	50	--	--	--	50
Mercury	2	--	--	--	2	--
ORGANICS						
Chloroform	100[c]	--	--	--	100	--
Tetrachloroethylene	--	--	5	--	5	--
Toluene	--	--	2,000	40	--	--
Trichloroethylene	5	--	--	--	5	--

[a]Federal Register, May 22, 1989
[b]Proposed in Federal Register, August 18, 1988.
[c]for total trihalomethanes; refers to the sum of chloroform, dibromochloromethane, bromodichloromethane and bromoform.

TABLE 5-3
OTHER GUIDANCE VALUES TO BE CONSIDERED
FOR CHEMICALS OF CONCERN IN GROUNDWATER
WDI SITE

Chemical	California AALs[a] (μg/liter)	Existing Federal MCLGs (μg/liter)	Proposed Federal MCLGs (μg/liter)[b]
INORGANICS			
Arsenic	--	--	--
Lead	--	--	0[c]
Manganese	--	--	--
Mercury	2	--	2
ORGANICS			
Chloroform	6	--	--
Tetrachloroethylene	--	--	0
Toluene	100	--	2,000
Trichloroethylene	7	0	--

[a] AALs as of 12/15/88.
[b] Proposed in Federal Register, May 22, 1989.
[c] Proposed in Federal Register, August 18, 1988.

Contaminants for which ambient air standards are available include lead and vinyl chloride. These standards are presented in Table 5-4. Exposure point concentrations of vinyl chloride presented in Table 4-7 were compared to the California AAQS. All exposure point concentrations were less than the ambient air standard. The estimated exposure point concentrations of lead are all below the NAAQS. Other potential guidance and criteria include the California AALs for air; these are presented in Table 5-5. Exposure point concentrations for all potential air contaminants are below regulatory guidance values.

5.2 RISK CHARACTERIZATION

For those contaminants where ARARs are not available for all contaminants, a quantitative risk assessment is performed to evaluate medium specific risks. For the WDI site, no soil ARARs were available; groundwater and air ARARs are not available for all of the chemicals of potential concern.

To quantitatively assess the potential risks to human health associated with the current-use and future-use exposure scenarios considered in this assessment, the concentrations of chemicals in relevant environmental media at points of potential exposure (exposure point concentrations) were developed in Section 4.3 and are then converted to chronic daily intakes (CDI) in this section. CDIs are expressed as the amount of a substance taken into the body per unit body weight per unit time, or mg/kg/day. A CDI is averaged over a lifetime for carcinogens, and over the exposure period for noncarcinogens (EPA, 1986b).

For recognized and/or potential carcinogens, excess lifetime cancer risks are obtained by multiplying the chronic daily intake (CDI) of the contaminant under consideration by its cancer potency factor (q^*). This is appropriate for cancer risks of less than 10^{-2} (i.e., 1 excess cancer in every 100 individuals exposed throughout their lifetime). When the daily intakes are large, the linear approach described above is not valid. For excess

TABLE 5-4
APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARS)
FOR CHEMICALS OF CONCERN IN AIR
WDI SITE

Chemical	National AAQS ($\mu g/m^3$)	California AAQS ($\mu g/m^3$)
Volatile Organics		
Benzene[a]	--	--
Carbon Tetrachloride	--	--
Chloroform	--	--
1,2-Dibromoethane	--	--
1,2-Dichloroethane	--	--
Tetrachloroethylene	--	--
1,1,1-Trichloroethane	--	--
Trichloroethylene	--	--
Vinyl Chloride[a]	--	26
Particulates[b]		
Lead	1.5	1.5

[a] Fugitive emissions should be contained as required under National Emission Standards for Hazardous Air Pollutants (NESHAPs) of the Clean Air Act.

[b] No air standards available for particulate contaminants except lead.

TABLE 5-5

GUIDANCE VALUES TO BE CONSIDERED
FOR CHEMICALS OF CONCERN IN AMBIENT AIR
AT THE WASTE DISPOSAL, INC. SITE

Chemical	California Air AALs[a] ($\mu g/m^3$)
Benzene	3.2
Carbon Tetrachloride	--
Chloroform	0.6
1,2-Dibromoethane	--
1,2-Dichloroethane	--
Mercury	0.07
PAHs-noncarcinogenic	1.9
Tetrachloroethylene	--
1,1,1-Trichloroethane	310
Trichloroethylene	7
Vinyl chloride	--

[a] California AALs as of 12/15/88.

lifetime cancer risks greater than 10^{-2}, individual excess lifetime cancer risks are calculated by:

$$\text{Excess Lifetime Cancer Risk} = 1 - \exp(-\text{CDI} \times q^*)$$

In general, EPA cancer potency factors based on animal data represent the 95-percent upper-confidence limit values based on a linearized multistage model. Thus, the actual risks associated with exposure to a potential carcinogen quantitatively evaluated based on animal data area not likely to exceed the risks estimated using these cancer potency factors. However, they may be lower. EPA cancer potencies based on human epidemiological data (e.g., arsenic) are point estimates based on a linear absolute risk model. In its Health Assessment Document for Arsenic (EPA, 1984m), the Agency notes that "while it is unlikely that the true risks would be higher than these estimates, they could be substantially lower". Since more than one potentially carcinogenic chemical is present at the WDI site, the total potential risk will be estimated by summing the potential risks for individual chemicals, as suggested in EPA's guidance document for chemical mixtures (EPA, 1986c).

Potential risks are assessed for noncarcinogens by the ratio of the chronic daily intake (CDI) exposure to the reference dose (CDI:RfD). In general, if the CDI:RfD ratio is less than one (i.e., if the daily intake is below the designated EPA health criterion), the contaminant is considered unlikely to be associated with any significant health risks and is, therefore, less likely to be of regulatory concern than a CDI:RfD ratio greater than a ratio on one.

Toxic effects of noncarcinogenic chemicals are initially assumed to be additive, in accordance with EPA guidance on health risk assessment of complex mixtures (EPA, 1986c). For each scenario, the CDI:RfD ratios for each individual chemical are summed to produce a Hazard Index (HI) for total noncarcinogenic risk. If the Hazard Index is less than one, the combined intake of chemicals by the exposure route under consideration is unlikely to

pose a health risk. If the Hazard Index exceeds one, the chemicals are subdivided according to their toxicological effects (critical endpoints), and the risk for each endpoint is considered separately (EPA, 1986c).

In this section of the risk assessment document, the biological intakes of chemicals of potential concern by potentially exposed populations are calculated. To determine these potential intakes, assumptions are made concerning chemical concentrations, environmental fate and transport, exposed populations, and exposure conditions, such as frequency and duration of exposure. According to current EPA guidance (EPA, 1988h) for each exposure scenario evaluated there are two exposure cases: 1) an average exposure case where mean concentrations are used, together with what are considered to be the most likely exposure conditions, and 2) a plausible maximum case where, generally the highest measured concentrations are used, together with upper range estimates of potential exposure parameters relating to the frequency and duration of exposure, as well as the quantity of contaminated media contacted. It should be noted that the exposure assumptions used for the plausible maximum case, while considered possible, are realistically likely only to apply to a very small segment of the potentially exposed populations.

In the following sections, potential human health risks are evaluated for all complete exposure pathways under both current and future use conditions. Assumptions and procedures used to calculate these values are discussed below for each exposure scenario that is quantitatively evaluated. Sample calcuations for each exposure route are included in Appendix C.

5.2.1 CURRENT-USE CONDITIONS

Under the current-use conditions, individuals could be exposed to site related contaminants via direct contact with contaminated soils and inhalation of airborne particulates and subsurface gas.

5.2.1.1 Direct Contact with Soil

As discussed in Section 4.3.1, direct contact with contaminated soil can lead to exposure via inadvertent or intentional soil ingestion via hand-to-mouth contact, deposition in the upper respiratory tract of inhaled particles which are subsequently swallowed, or dermal absorption.

Students from the high school may trespass on the WDI site coming in contact with on-site soil contaminants. For purposes of this assessment, trespassers on the WDI site are assumed to be high school students who trespass once a week under average conditions and five times a week under plausible maximum conditions, throughout the year.

In estimating risks over the exposure period, it was assumed that concentrations of the chemicals of concern in surface soils will remain constant over the period of exposure. This assumption is reasonable for those chemicals of potential concern which tend to persist in soil; it may lead to overestimation of exposure for monocyclic aromatic hydrocarbons and the lower-molecular weight PAHs, which are less strongly sorbed to soil particles and may slowly volatilize.

The amount of soil adhering to skin was assumed to be 1.45 mg/cm^2 under average conditions and 2.77 mg/cm^2 under for plausible maximum conditions based on information presented in the EPA Superfund Exposure Assessment Manual (1988e). The area of skin contacting soil (cm^2) was derived from a study by EPA (1985r), and was based on soil contacting both hands and forearms of teenagers.

Absorption through intact skin of the inorganic chemicals of concern is assumed to be negligible for all chemicals except mercury. Mercury and the organic chemicals are assumed to be absorbed through the skin to some extent. However, the extent of dermal absorption for chemicals present in a soil matrix has not been well studied and is difficult to quantify. The derivation

of the dermal and oral absorption rates for organic chemicals of concern are detailed below.

Experimental data on the amount of chemicals that may be absorbed through the skin from soil under the environmental conditions assumed to occur for this assessment is limited. Factors that can affect the dermal absorption of a chemical include concentration in the applied dose, the site of exposure, inter-individual variability, and the vehicle in which the chemical is delivered to the skin (e.g., in a solvent matrix). For the purposes of this assessment, the amount of exposure due to dermal absorption is evaluated by estimating the fraction of absorption from contacted soil that may occur for each chemical of concern. Where relevant experimental data are available, they are used to derive absorption factors. Where such data are unavailable, absorption factors are assumed by analogy to other chemicals' affinities to be absorbed through the skin and their relative affinities for the water and lipid phases, and/or default value of 10%. Experimental data were used to derive the absorption factors for PAHs (Yang et al., 1986a,b), PCBs (Wester et al., 1987), organochlorine pesticides (Poiger and Schlatter, 1980), benzoic acid (Feldman and Maibach, 1970), phenolic compounds (Poiger and Schlatter, 1980). Except for mercury, dermal absorption of inorganics from contacted soil is assumed to be negligible (less than one percent) based on observations from animal studies using concentrated solutions (Skog and Wahlberg, 1964; Wahlberg, 1968a). Absorption of mercury is assumed to be greater than for the other inorganics based on reports of mercury absorption after topical application (Bourgeois et al., 1986) and the tendency of mercury to be present in liquid or vapor phases rather than a solid phase (Hursh et al., 1989). For the remaining chemicals, dermal absorption factors were assumed as described above.

Exposure to the chemical of concern also may occur as a result of inadvertent ingestion of soil during normal activities. Adults may ingest soil through swallowing dust particles, or through hand-to-mouth contact during eating or smoking. Based on EPA guidance, a soil ingestion rate of 100

mg/day for individuals older than 6 years will be used for the average and the plausible maximum cases (EPA, 1989c).

Relative bioavailability factors were also established for exposure resulting from incidental ingestion of soil to take into account the reduced bioavailability of the chemicals of potential concern from a soil matrix relative to their availability from a solvent or feed matrix. Bioavailability factors were derived from several studies on the gastrointestinal absorption of soil-adsorbed 2,3,7,8-tetrachloro-dibenzo-p-dioxin (TCDD) (Poiger and Schlatter, 1980; McConnell et al., 1984; Lucier et al., 1986). The experimental evidence indicates that TCDD is less readily absorbed through the gastrointestinal tract when adsorbed on soil. The fraction that became bioavailable was dependent in part on the composition of the soil or particulate matrix (e.g., amount of organic carbon) and the length of contact between the soil and the chemical. For pesticides, polycyclic aromatic hydrocarbons (PAHs), and polychlorinated biphenyls (PCBs) a reasonable assumption is that their adsorptive properties will approach those of TCDD because they have similar absorption potentials to soil as seen by the similarities in K_{oc}s. Consequently, the effect of a soil matrix on their gastrointestinal absorption is expected to be as described above. The bioavailability factors derived from the experimental studies (expressed as a relative percentage of administered dose) are 15% for the average case and 50% for the plausible maximum case for pesticides, PAHs, and PCBs. For most other organic chemicals, there is no information on the extent to which they may desorb from soil, and therefore, their bioavailability is conservatively assumed to be one (100%). Arsenic absorption from soil is assumed to be 80% for both the average and plausible maximum scenarios based on animal feed studies (EPA, 1984m). The bioavailability for the other inorganic compounds is assumed to be 100%.

The chemical intakes (ITKs) due to direct contact of the chemicals of concern present in surface soils were calculated as follows:

$$ITK_{dc} = ITK_i + ITK_d \qquad \text{(Equation 5-1)}$$

where,

$$ITK_i = (C)(BIO)(IN)(Y) \quad \text{(Equation 5-2)}$$

where,

ITK_i = chemical intake from ingestion per visit (mg/day),
C = chemical concentration (mg/kg),
IN = soil ingestion rate (mg/day),
Y = conversion factor (1 kg/10^6 mg), and
BIO = gastrointestinal absorption factor (unitless).

and,

$$ITK_d = (C)(ABS)(S)(SA)(Y) \quad \text{(Equation 5-3)}$$

where,

ITK_d = chemical intake from dermal absorption per visit (mg/day),
C = chemical concentration (mg/kg),
ABS = dermal absorption fraction,
S = soil contact rate (mg/cm^2/day),
SA = surface area (cm^2), and
Y = conversion factor (1 kg/10^6 mg).

The estimated chemical intake from the combined dermal and ingestion pathways was then converted to a chronic daily intake (CDI).

As discussed earlier in this section, the first step in quantification of human health risk is to calculate a chronic daily intake (CDI) for all contaminants. The CDI for the direct contact pathway is calculated using the following equation:

$$CDI_{dc} = [(ITK_{dc})(D)(F)]/[(BW)(E)(365)] \quad \text{(Equation 5-4)}$$

where,

CDI_{dc} = average daily intake via direct contact (mg/kg/day),
ITK_{dc} = chemical intake per visit (mg/day),
D = duration of exposure (years),
F = frequency of exposure (days/year),

E	-	extrapolation factor (years):
		for noncarcinogens - exposure period (years),
		for carcinogens - 75 year lifetime,
BW	-	body weight (kg), and
365	-	conversion factor (days/year).

On-site surface soil sampling results from the WDI site were used to estimate the exposure to trespassers. Geometric mean soil concentrations were used for the average case; maximum soil concentrations were used for the plausible maximum case.

In order to make the chronic daily intake (CDI) estimates comparable with cancer potency factors, which are based on chronic lifetime exposure, the CDIs for all carcinogenic chemicals are extrapolated over a 75-year average lifetime. The CDIs for noncarcinogenic chemicals are averaged over the estimated exposure period. For purposes of this assessment, lead will be considered a noncarcinogen. This assumption is currently under review by EPA and may be changed in the future.

Exposure assumptions for the current use direct contact pathway are listed in Table 5-6. Chronic daily intake estimates and risk estimates for the direct contact pathway under current-use conditions are presented in Table 5-7. Estimated cancer risks for trespassers on the WDI site are 5×10^{-7} under average conditions and 3×10^{-5} under plausible maximum conditions. The Hazard Index for the plausible maximum case was greater than one, due primarily to lead. The CDI:RfD ratio for lead was equal to one under plausible maximum conditions. However for all other chemicals, all individual CDI:RfD ratios were less than one for both average and plausible maximum cases.

TABLE 5-6
ASSUMPTIONS USED TO EVALUATE THE DIRECT CONTACT PATHWAY
UNDER CURRENT USE CONDITIONS
WDI SITE

Parameter	Average Case	Plausible Maximum Case
Frequency of Exposure	1 event/week	5 events/week
Age during exposure Period	14 - 17 years	13 - 18 years
Duration of Exposure	4 years	6 years
Average Weight over exposure period[a]	60 kg	60 kg
Area of Exposed Skin[a]	1400 cm^2	1980 cm^2
Rate of Incidental Soil Ingestion[b]	100 mg	100 mg
Lifetime	75 years	75 years
Soil Contact Rate[a]	1.45 $mg/cm^2/day$	2.77 $mg/cm^2/day$
Oral Absorption Factor		
Pesticides, PCBs, PAHs[c]	0.15	0.50
Arsenic[d]	0.80	0.80
All other Chemicals (including pentachlorophenol)	1	1
Dermal Absorption Factor		
Volatile Organic Chemicals[e]	0.1	0.1
Noncarcinogenic PAHs[c]	0.03	0.05
Carcinogenic PAHs[c,g]	0.009	0.02
PCBs[c]	0.07	0.07
Phenolic Compounds[h]	0.2	0.3
DDT[c]	0.02	0.02

TABLE 5-6 (cont.)

ASSUMPTIONS USED TO EVALUATE THE DIRECT CONTACT PATHWAY
UNDER CURRENT USE CONDITIONS
WDI SITE

Parameter	Average Case	Plausible Maximum Case
Dieldrin[c] and other chlorinated pesticides	0.01	0.02
Benzoic Acid[f]	0.36	0.36
Mercury	0.1	0.1
Other Inorganics	0	0

[a] EPA, 1985r.
[b] EPA, 1989c.
[c] Poiger and Schlatter, 1980.
[d] EPA, 1984m.
[e] Based on analogy to other chemicals.
[f] Feldman and Maibach, 1970.
[g] Yang et al., 1984; Wester et al., 1987.
[h] Roberts et al., 1977.
[i] Skog and Wahlberg, 1964.

TABLE 5-7
EXPOSURES AND RISKS ASSOCIATED WITH DIRECT CONTACT
TO SURFACE SOILS BY TRESPASSERS
WDI SITE
A. POTENTIAL CARCINOGENS

Chemical	Chronic Daily Intake CDI (mg/kg/day) Average Case	Chronic Daily Intake CDI (mg/kg/day) Plausible Maximum Case	Upperbound Excess Lifetime Cancer Risks Average Case	Upperbound Excess Lifetime Cancer Risks Plausible Maximum Case
INORGANICS				
Arsenic	8.1E-08	6.5E-06	2E-07	1E-05
ORGANICS				
Benzene	1.0E-08	1.6E-07	3E-10	5E-09
Chlordane	4.6E-11	3.2E-08	6E-11	4E-08
DDT	9.6E-10	6.0E-07	3E-10	2E-07
Dieldrin	1.7E-10	4.2E-08	3E-09	7E-07
Heptachlor Epoxide	4.3E-11	7.0E-09	4E-10	6E-08
Methylene Chloride	8.8E-10	3.7E-07	7E-12	3E-09
PAHs - Carcinogenic	3.7E-09	3.7E-07	4E-08	4E-06
PCBs	4.2E-08	2.0E-06	3E-07	2E-05
Pentachlorophenol	9.6E-09	1.8E-07	2E-10	3E-09
TOTAL			5E-07	3E-05

B. NONCARCINOGENS

Chemical	CDI (mg/kg/day) Average	CDI (mg/kg/day) Plausible Maximum	CDI:RfD Ratios Average	CDI:RfD Ratios Plausible Maximum
INORGANICS				
Arsenic	1.5E-06	8.1E-05	2E-03	8E-02
Antimony	1.3E-06	9.0E-06	3E-03	2E-02
Cadmium	2.6E-07	3.9E-06	3E-04	6E-01
Chromium	5.5E-06	6.3E-05	1E-03	2E-01
Copper	8.5E-06	6.1E-04	2E-04	2E-02
Lead	1.0E-05	8.7E-04	2E-02	1E+00
Manganese	8.6E-05	9.2E-06	4E-04	5E-05
Mercury	1.1E-07	9.2E-06	4E-04	3E-02
Selenium	1.2E-07	1.4E-06	4E-05	5E-04
Thallium	1.9E-06	2.2E-05	3E-02	3E-01
ORGANICS				
2-Butanone	5.9E-09	3.5E-07	1E-07	7E-06
Benzoic Acid	3.0E-07	5.7E-06	7E-08	1E-06
Chlordane	8.6E-10	4.0E-07	1E-05	7E-03
DDT	1.8E-08	7.5E-06	4E-05	1E-02
Dieldrin	3.1E-09	5.3E-07	6E-05	1E-02
Ethylbenzene	3.4E-08	1.2E-05	3E-07	1E-04
Heptachlor Epoxide	8.0E-10	8.7E-08	6E-05	7E-03
Methylene Chloride	1.7E-08	7.0E-05	3E-07	1E-03
PAHs- Noncarcinogenic	7.6E-07	7.0E-05	2E-06	2E-04
Pentachlorophenol	8.2E-08	9.1E-07	3E-06	3E-05
Toluene	3.7E-08	6.9E-05	1E-07	2E-04
Xylenes	1.3E-07	1.9E-05	7E-08	1E-05
HAZARD INDEX			5E-02	3E+00

5.2.1.2 Inhalation of Airborne Particulate

Individuals living or attending school may be exposed to airborne particulates generated by wind erosion of surface soils. Since ambient air data were not available, the exposure point concentrations were generated based on surface soil concentrations as discussed in Section 4.3. Geometric mean surface soil concentrations were used to estimate airborne particulate for both average and plausible maximum scenarios.

In evaluating exposure, area residents are assumed to be exposed 330 days per year, 24 hours/day, for 9 or 30 years under average and plausible maximum case conditions, respectively. The exposure frequency takes into account the number of days with precipitation greater than 0.01 inch, on which dust generation is expected to be minimal (NOAA, 1983). Risks to area residents will be quantified for residents living within 0.1, 0.5, and 1 km of the WDI site. A respiration rate of 20 m^3/day was used to evaluate both average and plausible maximum conditions (EPA, 1988a). Indoor air concentrations were assumed to be equal to the outside air concentrations.

Students are assumed to be exposed to airborne contaminants 180 days per year for 4 years under the average case and 6 years under the plausible maximum case. Duration of exposure is assumed to be 8 hours/day under average conditions and 10 hours/day under plausible maximum conditions. Students are anticipated to be exposed at 0.1 km from the WDI site. An inhalation rate of 20 m^3/day was assumed for both the average and plausible maximum cases. The assumptions used in this scenario are presented in Table 5-8.

Absorption by inhalation of all contaminants of concern but arsenic is estimated to be 100% for both the average and the plausible maximum exposure cases. For arsenic, a 30% absorption rate has been assumed in deriving the inhalation cancer potency factor and will be used in this exposure assessment (EPA, 1984m).

TABLE 5-8
ASSUMPTIONS USED TO EVALUATE THE INHALATION PATHWAYS
UNDER CURRENT USE CONDITIONS
WDI SITE

Parameter	Average Case	Plausible Maximum Case
Off-site Adult Residents		
Frequency of Exposure	330 days/year	330 days/year
Duration of Exposure	24 hours/day	24 hours/day
Exposure Period	9 years	30 years
Age during Exposure Period	Adult	Adult
Average Weight over Exposure Period[a]	70 kg	70 kg
Inhalation Rate[b]	20 m^3/day	20 m^3/day
Students		
Frequency of Exposure	180 days/year	180 days/year
Duration of Exposure	8 hours/day	10 hours/day
Exposure Period	4 years	6 years
Age during Exposure Period	14 - 17 years	13 - 18 years
Average Weight over Exposure Period[a]	60 kg	60 kg
Inhalation Rate[c]	20 m^3/day	20 m^3/day
General		
Lifetime	75 years	75 years
Inhalation Absorption Factor		
Arsenic[d]	0.3	0.3
All Other Chemicals	1	1

[a] EPA, 1985r.
[b] EPA, 1988a.
[c] EPA, 1988a.
[d] EPA, 1984m.

The estimated intakes of contaminants absorbed via inhalation exposure were calculated using the following equation:

$$ITK_a = (C)(I)(L)(A)(Y) \qquad \text{(Equation 5-5)}$$

where,

- ITK_a - chemical intake over the duration of exposure (mg/day);
- C - chemical concentration in air (mg/m^3);
- I - respiration rate (m^3/day);
- L - length of exposure (hrs/day);
- A - inhalation absorption factor (unitless); and
- Y - conversion factor (1 day/24 hours).

The chemical intake was converted to a chronic daily intake as follows:

$$CDI_a = ITK_a(D)(F)/[(BW)(E)(365)] \qquad \text{(Equation 5-6)}$$

where,

- CDI_a - chronic daily intake (mg/kg/day);
- ITK_a - chemical intake via air (mg);
- D - exposure period (years);
- F - frequency of exposure (days/year);
- BW - body weight (kg);
- E - extrapolation factor:
  - for noncarcinogens - exposure period,
  - for carcinogens - 75 year lifetime; and
- 365 - exposure conversion factor (days/year).

Average and maximum plausible chronic daily intake and risk estimates for exposure to airborne contaminants are presented in Table 5-9 for residents. Cancer risks estimated for current residents downwind of the WDI site ranged from 2×10^{-7} to 3×10^{-6} for average conditions and 8×10^{-7} to 8×10^{-6} for plausible maximum conditions. Risks were higher for individuals closer to the WDI site. The CDI:RfD ratios for noncarcinogenic endpoints are all less than one; the Hazard Indices are also less than one.

The risk estimates presented above are based on constant wind direction over the exposure period. In order to scale these values for potential risk for residents living in a certain direction from the site, the wind rose data presented in Section 2 will be used to apportion the risk over four quadrants. For individuals residing northeast of the WDI site, the cancer risk values are 38% of the calculated value; therefore, under average conditions, the cancer risk ranges from 8×10^{-8} to 1×10^{-6}, and under plausible maximum conditions the cancer risk ranges from 3×10^{-7} to 3×10^{-6}. The overall cancer risks for other quadrants will be lower, since the winds at the WDI site blow predominantly from the Southwest. The Hazard Index remains less than one after adjusting for wind direction.

The CDI and risk estimates for students are listed in Table 5-10. For students, the cancer risk estimates are 2×10^{-7} under average conditions and 4×10^{-7} under plausible maximum conditions. The CDI:RfD ratios are less than one for all chemicals of concern; the Hazard Indices are also less than one for both average and plausible maximum cases indicating a low potential for adverse health effects.

St. Paul's High School is located northeast of the WDI site. As discussed above, the wind blows in that direction approximately 38 percent of the time. The cancer risk values range from 8×10^{-8} under average conditions to 2×10^{-7} under plausible maximum conditions, after adjusting for wind direction variation.

5.2.1.3 Inhalation of Volatiles from Subsurface Gas

As discussed in Section 4.3, individuals working, living, or attending school downwind from the WDI site may be exposed to subsurface gas contaminants which have migrated to the surface and been released to ambient air. Exposure point concentrations used to evaluate this pathway are discussed in Section 4.3.1.4 and presented in Table 4-7.

TABLE 5-9

EXPOSURES AND RISKS ASSOCIATED WITH INHALATION OF PARTICULATES BY CURRENT RESIDENTS LIVING NEAR THE WDI SITE

A. POTENTIAL CARCINOGENS

	Chronic Daily Intake (mg/kg/day)						Upperbound Excess Lifetime Cancer Risks					
	for 0.1 km		for 0.5 km		for 1.0 km		for 0.1 km		for 0.5 km		for 1.0 km	
Chemical	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case
INORGANICS												
Arsenic	5.6E-09	1.9E-08	1.0E-09	3.4E-09	5.4E-10	1.8E-09	3E-07	9E-07	5E-08	2E-07	3E-08	9E-08
Cadmium	2.5E-09	8.4E-09	4.6E-10	1.5E-09	2.4E-10	8.2E-10	2E-08	5E-08	3E-09	9E-09	1E-09	5E-09
Chromium	5.3E-08	1.8E-07	9.9E-09	3.3E-08	5.3E-09	1.8E-08	2E-06	7E-06	4E-07	1E-06	2E-07	7E-07
ORGANICS												
Benzene	5.9E-10	2.0E-09	1.1E-10	3.7E-10	5.9E-11	2.0E-10	2E-11	6E-11	3E-12	1E-11	2E-12	6E-12
Chlordane	2.3E-11	7.6E-11	4.3E-12	1.4E-11	2.3E-12	7.5E-12	3E-11	1E-10	6E-12	2E-11	3E-12	1E-11
DDT	3.1E-10	1.0E-09	5.9E-11	2.0E-10	3.1E-11	1.0E-10	1E-10	4E-10	2E-11	7E-11	1E-11	4E-11
Dieldrin	8.7E-11	2.9E-10	1.6E-11	5.5E-11	8.7E-12	2.9E-11	1E-09	5E-09	3E-10	9E-10	1E-10	5E-10
Heptachlor Epoxide	2.2E-11	7.3E-11	4.0E-12	1.3E-11	2.2E-12	7.2E-12	2E-10	7E-10	4E-11	1E-10	2E-11	7E-11
Methylene Chloride	5.3E-11	1.8E-10	9.9E-12	3.3E-11	5.3E-12	1.8E-11	7E-13	2E-12	1E-13	5E-13	7E-14	2E-13
PAHs	2.0E-09	6.8E-09	3.7E-10	1.2E-09	2.0E-10	6.7E-10	1E-08	4E-08	2E-09	8E-09	1E-09	4E-09
PCBs	5.0E-09	1.7E-08	9.0E-10	3.0E-09	4.6E-10	1.5E-09	4E-08	1E-07	7E-09	2E-08	4E-09	1E-08
Pentachlorophenol	5.6E-10	1.9E-09	1.1E-10	3.5E-10	5.6E-11	1.9E-10	9E-12	3E-11	2E-12	6E-12	9E-13	3E-12
TOTAL							3E-06	8E-06	5E-07	2E-06	2E-07	8E-07

TABLE 5-9 -CONTINUED-
EXPOSURES AND RISKS ASSOCIATED WITH INHALATION OF PARTICULATES BY CURRENT RESIDENTS
WDI SITE

B. NONCARCINOGENS

Chemical	Chronic Daily Intake (mg/kg/day)						CDI:RfD Ratios					
	for 0.1 km		for 0.5 km		for 1.0 km		for 0.1 km		for 0.5 km		for 1.0 km	
	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case
INORGANICS												
Antimony	1.0E-07	1.0E-07	1.9E-08	1.9E-08	1.0E-08	1.0E-08	3E-04	3E-04	5E-05	5E-05	3E-05	3E-05
Copper	7.0E-07	7.0E-07	1.3E-07	1.3E-07	6.7E-08	6.7E-08	7E-05	7E-05	1E-05	1E-05	7E-06	7E-06
Lead	8.3E-07	8.3E-07	1.5E-07	1.5E-07	7.7E-08	7.7E-08	1E-03	1E-03	2E-04	2E-04	1E-04	1E-04
Mercury	2.8E-09	2.8E-09	5.2E-10	5.2E-10	2.6E-10	2.6E-10	6E-05	6E-05	1E-05	1E-05	5E-06	5E-06
Selenium	9.6E-09	9.6E-09	1.6E-09	1.6E-09	8.5E-10	8.5E-10	1E-05	1E-05	2E-06	2E-06	9E-07	9E-07
Thallium (a)												
ORGANICS												
2-Butanone	1.6E-10	1.6E-10	2.8E-11	2.8E-11	1.5E-11	1.5E-11	2E-09	2E-09	3E-10	3E-10	2E-10	2E-10
Ethylbenzene	9.0E-10	9.0E-10	1.7E-10	1.7E-10	8.8E-11	8.8E-11	9E-09	9E-09	2E-09	2E-09	9E-10	9E-10
Pentachlorophenol	4.6E-09	4.6E-09	8.8E-10	8.8E-10	4.6E-10	4.6E-10	2E-07	2E-07	3E-08	3E-08	2E-08	2E-08
PAHs - Noncarcinogenic	8.5E-08	8.5E-08	1.6E-08	1.6E-08	8.3E-09	8.3E-09	2E-07	2E-07	4E-08	4E-08	2E-08	2E-08
Toluene	1.0E-09	1.0E-09	1.9E-10	1.9E-10	9.8E-11	9.8E-11	1E-09	1E-09	2E-10	2E-10	1E-10	1E-10
Xylenes	3.6E-09	3.6E-09	7.0E-10	7.0E-10	3.6E-10	3.6E-10	1E-08	1E-08	2E-09	2E-09	1E-09	1E-09
HAZARD INDEX							2E-03	2E-03	3E-04	3E-04	2E-04	2E-04

(a) Risks associated with thallium were not quantified for inhalation pathways, because no appropriate toxicity value was available.

TABLE 5-10

EXPOSURES AND RISKS ASSOCIATED WITH INHALATION OF PARTICULATES ORIGINATING FROM SURFACE SOILS BY STUDENTS 0.1 km FROM THE WDI SITE

A. POTENTIAL CARCINOGENS

Chemical	Chronic Daily Intake (CDI) (mg/kg/day)		Upperbound Excess Lifetime Cancer Risks	
	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case
INORGANICS				
Arsenic	5.3E-10	9.9E-10	3E-08	5E-08
Cadmium	2.4E-10	4.4E-10	1E-09	3E-09
Chromium	5.0E-09	9.3E-09	2E-07	4E-07
ORGANICS				
Benzene	5.6E-11	1.0E-10	7E-11	3E-12
Chlordane	2.2E-12	4.1E-12	3E-12	5E-12
DDT	2.9E-11	5.5E-11	1E-11	2E-11
Dieldrin	8.2E-12	1.5E-11	1E-10	2E-10
Heptachlor Epoxide	2.1E-12	3.9E-12	2E-11	4E-11
Methylene Chloride	5.0E-12	3.9E-12	3E-11	5E-14
PAHs	1.9E-10	3.6E-10	1E-09	2E-09
PCBs	4.7E-10	8.8E-10	4E-09	7E-09
Pentachlorophenol	5.3E-11	9.9E-11	4E-10	2E-12
TOTAL			2E-07	4E-07

B. NONCARCINOGENS

Chemical	Chronic Daily Intake (CDI) (mg/kg/day)		Cancer Risks	
	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case
INORGANICS				
Antimony	2.2E-08	2.7E-08	5E-05	7E-05
Copper	1.5E-07	1.8E-07	1E-05	2E-05
Lead	1.8E-07	2.2E-07	3E-04	4E-04
Mercury	6.0E-10	7.5E-10	1E-05	1E-05
Selenium	2.0E-09	2.5E-09	2E-06	3E-06
Thallium (a)				
ORGANICS				
2-Butanone	3.3E-11	4.2E-11	4E-10	5E-10
Ethylbenzene	1.9E-10	2.4E-10	2E-09	2E-09
Pentachlorophenol	9.9E-10	1.2E-09	3E-08	4E-08
PAHs - Noncarcinogenic	1.8E-08	2.3E-08	4E-08	6E-08
Toluene	2.1E-10	2.7E-10	2E-10	3E-10
Xylenes	7.7E-10	9.6E-10	3E-09	3E-09
HAZARD INDEX			4E-04	5E-04

(a) Risks for thallium were not quantified for inhalation pathways.

The estimated intakes of volatile contaminants absorbed via inhalation exposure were estimated using Equation 5-5 presented in Section 5.2.1.2. The chronic daily intake values were calculated using Equation 5-6. Exposure assumptions are listed in Table 5-8. Exposure is assumed to occur 330 days per year for residents and 180 days per year for students. Average and plausible maximum chronic daily intakes and risk estimates for exposure to subsurface gas contaminants under current-use conditions are presented in Table 5-11 for residents. The overall cancer risks for residents ranged from 2×10^{-8} to 3×10^{-7} under average conditions and from 5×10^{-7} to 5×10^{-6} under plausible maximum conditions. The CDI:RfD ratio for residents was less than one in all scenarios.

As discussed in Section 5.2.1.2, these inhalation risk estimates assume constant wind direction. For residents living northeast of the WDI site, the risks are adjusted by a factor of 0.38, resulting in 8×10^{-9} to 1×10^{-7} risks under average case conditions and 2×10^{-7} to 2×10^{-6} under plausible maximum conditions. Inhalation risks to off-site residents living in other wind directions would have risks lower than those listed above.

Chronic daily intake and risk estimates for students are presented in Table 5-12. For students, the upperbound cancer risk estimates were 3×10^{-8} for the average case and 3×10^{-7} for the plausible maximum case. When these risk estimates are adjusted for wind direction, the resulting upperbound cancer risks are 1×10^{-8} for average case conditions and 1×10^{-7} for plausible maximum conditions. The CDI:RfD ratios for both average and plausible maximum conditions were less than one for 1,1,1-trichloroethane, the only chemical with a noncarcinogenic endpoint via inhalation.

5.2.2 FUTURE USE CONDITIONS

Under future-use scenarios, the WDI site may be developed for residential purposes allowing increased access to the site and its contaminants. The future-use scenarios to be quantified are:

TABLE 5-11

EXPOSURES AND RISKS ASSOCIATED WITH THE INHALATION
OF VOLATILE CHEMICALS EMITTED FROM SUBSURFACE GAS
BY RESIDENTS NEAR THE WDI SITE

A. POTENTIAL CARCINOGENS

Chemical	Chronic Daily Intake (mg/kg/day)						Upperbound Excess Lifetime Cancer Risks					
	for 0.1 km		for 0.5 km		for 1.0 km		for 0.1 km		for 0.5 km		for 1.0 km	
	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case
Benzene	1.9E-07	5.3E-06	3.4E-08	9.9E-07	1.8E-08	5.2E-07	5E-09	2E-07	1E-09	3E-08	5E-10	1E-08
Carbon Tetrachloride	2.9E-09	8.9E-08	5.6E-10	1.7E-08	2.9E-10	8.7E-09	4E-10	1E-08	7E-11	2E-09	4E-11	1E-09
Chloroform	7.4E-09	4.4E-07	1.4E-09	8.4E-08	7.1E-10	4.3E-08	6E-10	4E-08	1E-10	7E-09	6E-11	4E-09
1,2-Dibromoethane	3.0E-07	2.1E-06	5.6E-08	3.9E-07	2.9E-08	2.1E-07	2E-07	2E-06	4E-08	3E-07	2E-08	2E-07
1,2-Dichloroethane	8.7E-08	1.3E-06	1.6E-08	2.5E-07	8.4E-09	1.2E-07	8E-09	1E-07	1E-09	2E-08	8E-10	1E-08
Tetrachloroethylene	1.4E-07	7.6E-07	2.6E-08	1.4E-07	1.4E-08	7.5E-08	5E-10	3E-09	9E-11	5E-10	5E-11	2E-10
Trichloroethylene	2.6E-07	2.1E-06	5.0E-08	3.8E-07	2.6E-08	2.0E-07	3E-09	3E-08	6E-10	5E-09	3E-10	3E-09
Vinyl Chloride	8.7E-08	1.1E-05	1.6E-08	2.2E-06	8.7E-09	1.1E-06	3E-08	3E-06	2E-10	6E-07	1E-10	3E-07
TOTAL							3E-07	5E-06	5E-08	1E-06	2E-08	5E-07

B. NONCARCINOGENS

Chemical	Chronic Daily Intake (mg/kg/day)						CDI:RfD Ratios					
	for 0.1 km		for 0.5 km		for 1.0 km		for 0.1 km		for 0.5 km		for 1.0 km	
	Average Case	Maximum Case	Average Case	Maximum Case	Average Case	Maximum Case	Average Case	Maximum Case	Average Case	Maximum Case	Average Case	Maximum Case
1,1,1-Trichloroethane	6.2E-07	2.8E-06	1.2E-07	5.4E-07	6.2E-08	2.8E-07	2E-06	9E-06	4E-07	2E-06	2E-07	9E-07
HAZARD INDEX							2E-06	9E-06	4E-07	2E-06	2E-07	9E-07

TABLE 5-12
EXPOSURES AND RISKS ASSOCIATED WITH THE INHALATION OF
VOLATILES EMITTED FROM SUBSURFACE GAS BY STUDENTS

WDI SITE

A. POTENTIAL CARCINOGENS

Chemical	CDI (mg/kg/day) for 0.1 km		Upperbound Excess Lifetime Cancer Risks for 0.1 km	
	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case
Benzene	1.8E-08	2.8E-07	5E-10	8E-09
Carbon Tetrachloride	2.8E-10	4.6E-09	4E-11	6E-10
Chloroform	7.0E-10	2.5E-08	6E-11	2E-09
1,2-Dibromoethane	2.8E-08	1.1E-07	2E-08	8E-08
1,2-Dichloroethane	8.2E-09	7.2E-08	7E-10	7E-09
Tetrachloroethylene	1.3E-08	4.0E-08	4E-11	1E-10
Trichloroethylene	2.5E-08	1.1E-07	3E-10	1E-09
Vinyl Chloride	8.2E-09	6.0E-07	2E-09	2E-07
TOTAL			3E-08	3E-07

B. NONCARCINOGENS

Chemical	CDI (mg/kg/day) for 0.1 km		CDI:RfD Ratios for 0.1 km	
	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case
1,1,1-Trichloroethane	1.3E-07	8.2E-07	4E-07	3E-06
HAZARD INDEX			4E-07	3E-06

- direct contact with soil contaminants by on-site residents of three age groups,
- ingestion of contaminated groundwater, and
- inhalation of subsurface gas by on-site residents as a result of gas infiltration into homes.

5.2.2.1 Direct Contact with On-site Soils

On-site residents may be exposed to surface soil contaminants while playing or gardening. Since children under six have greater exposure to soil contaminants due to their high soil ingestion rates, exposure to two future resident populations will be evaluated: children aged 1 to 6, and adult residents. Exposure assumptions are listed on Table 5-13. Intake estimates were calculated using Equation 5-1; chronic daily intakes were estimated using Equation 5-2 discussed under current use conditions.

On-site adult residents are assumed to be exposed 240 and 365 days/year for 9 and 30 years under average and plausible maximum cases, respectively. Children aged one to six are assumed to reside at the site for six years under both average and plausible maximum conditions.

For adults, the exposed skin area is assumed to be 1400 cm^2 under average conditions and 1980 cm^2 under plausible maximum conditions, based on values presented by EPA (1985r, 1988f). For children, the exposed skin area will be assumed to be 1400 cm^2 in both average and plausible maximum conditions. A soil contact rate of 1.45 mg/cm^2 was assumed for average conditions and 2.77 mg/cm^2 for plausible maximum conditions for both adult and child scenarios. The dermal absorption factors discussed in Section 5.2.1.1 are used here.

A soil ingestion rate of 100 mg/day was assumed to estimate exposure to adults under both average and plausible maximum conditions. For children, a soil ingestion rate of 200 mg/day was assumed for the average case and 800 mg/day for the plausible maximum case to assess risks to a child with pica (excessive dirt consumption), based on EPA (1988f, 1989c) recommended values.

TABLE 5-13
ASSUMPTIONS USED TO EVALUATE THE DIRECT CONTACT
PATHWAY UNDER FUTURE-USE CONDITIONS
WDI SITE

Parameter	Average Case	Plausible Maximum Case
<u>On-site Adult Residents</u>		
Frequency of Exposure	240 days/year	365 days/year
Duration of Exposure[a]	9 years	30 years
Age during Exposure Period	Adult	Adult
Average Weight over Exposure Period[a]	70 kg	70 kg
Area of exposed skin[a]	1400 cm^2	1980 cm^2
Rate of Incidental Ingestion[b]	100 mg/event	100 mg/event
<u>On-site Child Residents (Aged 1-6 years)</u>		
Frequency of Exposure	240 days/year	365 days/year
Duration of Exposure[a]	6 years	6 years
Age During Exposure Period[a]	1-6 years	1-6 years
Average Weight over Exposure Period[a]	15 kg	15 kg
Area of Exposed Skin[a]	1400 cm^2	1400 cm^2
Rate of Incidental Ingestion[b]	200 mg/day	800 mg/day

TABLE 5-13 (cont.)

ASSUMPTIONS USED TO EVALUATE THE DIRECT CONTACT PATHWAY UNDER FUTURE-USE CONDITIONS
WDI SITE

Parameter	Average Case	Plausible Maximum Case
General		
Lifetime[a]	75 years	75 years
Oral Absorption Factor		
Pesticides, PCBs, PAHs[c]	0.15	0.50
Arsenic[d]	0.80	0.80
All other Chemicals (including pentachlorophenol)	1	1
Dermal Absorption Factor		
Benzoic Acid[g]	0.36	0.36
Phenolic Compounds[h]	0.02	0.03
DDT[c]	0.02	0.02
Other Chlorinated Pesticides[c]	0.01	0.02
Carcinogenic PAHs[e,c]	0.009	0.02
Noncarcinogenic PAHs[e]	0.03	0.05
PCBs[e]	0.07	0.07
Other organics[f]	0.1	0.1
Mercury[f]	0.1	0.1
Other inorganics[f]	0	0

TABLE 5-13 (cont.)

ASSUMPTIONS USED TO EVALUATE THE DIRECT CONTACT
PATHWAY UNDER FUTURE-USE CONDITIONS
WDI SITE

Parameter	Average Case	Plausible Maximum Case
Soil Contact Rate[a]	1.45 $mg/cm^2/day$	2.77 $mg/cm^2/day$

[a] EPA, 1988a.
[b] EPA, 1989c.
[c] Poiger and Schlatter, 1980.
[d] EPA, 1984m.
[e] Yang et al., 1984; Wester et al., 1987.
[f] Assumed Value.
[g] Feldman and Maibach, 1970.
[h] Roberts et al., 1977.

Oral absorption factors were assumed to be 15% and 50% for organochlorine pesticides, PAHs, and PCBs under the average and plausible maximum cases. Arsenic absorption from ingested soil was assumed to be 80% for both average and plausible maximum cases as discussed in Section 5.2.1.1. All other chemicals were assumed to be absorbed 100 percent from soil.

Chronic daily intake and risk estimates for on-site adult residents are presented in Table 5-14. Estimated upperbound cancer risks for on-site residents exposed via direct contact with surface soils range from 3×10^{-6} for the average case to 7×10^{-4} for the plausible maximum case. The Hazard Index for the average case is less than one. The Hazard Index for the plausible maximum exposure scenario is greater than one due primarily to lead, zinc, and DDT, which each had CDI:RfD ratios greater than one.

CDI and risk estimates for children aged one to six are presented in Table 5-15. Estimated upperbound cancer risks for young children range from 2×10^{-5} for the average case to 3×10^{-3} for the plausible maximum case. The Hazard Indices under both average and plausible maximum conditions exceed one. The CDI:RfD ratio for thallium exceeded one under average and plausible maximum conditions. The CDI:RfD ratios for arsenic, antimony, cadmium, chromium, copper, lead, mercury, DDT, chlordane, thallium, and zinc were greater than or equal to one under plausible maximum conditions.

5.2.3.2 Ingestion of Groundwater

Under future-use conditions, on-site residents may install a well for domestic use in the shallow aquifer underlying the WDI site. If this were to occur, adult residents could be exposed to groundwater contaminants on a daily basis for 9 years under average conditions or 30 years under plausible maximum conditions. Residents are assumed to consume 2 liters of water per day under both average and plausible maximum conditions (EPA, 1986a). Exposure assumptions are listed in Table 5-16. The estimated intakes of contaminants based on the ingestion of contaminated groundwater were calculated using the following equation:

TABLE 5-14
EXPOSURES AND RISKS ASSOCIATED WITH DIRECT CONTACT
TO SURFACE SOILS BY FUTURE ON-SITE RESIDENTS (ADULTS)
WDI SITE

A. POTENTIAL CARCINOGENS

Chemical	CDI (mg/kg/day)		Upperbound Excess Lifetime Cancer Risks	
	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case
INORGANICS				
Arsenic	5.9E-07	1.5E-04	1E-06	3E-04
ORGANICS				
Aldrin	9.2E-10	2.1E-08	2E-08	4E-07
Benzene	3.8E-08	7.0E-05	1E-09	2E-06
BHC (delta & gamma isomers)	1.9E-08	9.1E-07	3E-08	2E-06
Carbon Tetrachloride	6.8E-10	7.4E-09	9E-11	1E-09
Chlordane	3.1E-09	1.9E-06	4E-09	2E-06
Chloroform	5.5E-10	1.9E-08	3E-12	2E-06
DDT	1.3E-08	3.2E-04	4E-09	1E-04
Dieldrin	2.5E-09	2.6E-07	4E-08	4E-06
1,4-Dichlorobenzene	5.8E-08	8.9E-06	1E-09	2E-07
Heptachlor	3.5E-09	7.9E-08	2E-08	4E-07
Heptachlor Epoxide	3.8E-10	4.2E-08	3E-09	4E-07
Methylene Chloride	6.1E-09	4.4E-06	5E-11	3E-08
PAHs - Carcinogenic	5.0E-08	1.2E-05	6E-07	1E-04
PCBs	2.0E-07	1.4E-05	2E-06	1E-04
Pentachlorophenol	3.8E-08	4.8E-07	6E-10	8E-09
Tetrachloroethylene	9.6E-09	1.6E-04	5E-10	8E-06
Trichloroethylene	4.8E-08	1.9E-05	5E-10	2E-07
Vinyl Chloride	1.1E-08	6.3E-06	2E-08	1E-05
TOTAL			3E-06	7E-04

TABLE 5-14 (cont'd)

EXPOSURES AND RISKS ASSOCIATED WITH DIRECT CONTACT
TO SURFACE SOILS BY FUTURE ON-SITE RESIDENTS (ADULTS)

B. NONCARCINOGENS

Chemical	CDI (mg/kg/day)		CDI:RfD Ratios	
	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case
INORGANICS				
Arsenic	4.9E-06	3.9E-04	5E-03	4E-01
Antimony	4.9E-06	3.6E-05	1E-02	9E-02
Cadmium	9.4E-07	2.6E-05	9E-04	3E-02
Chromium	2.4E-05	2.1E-04	5E-03	4E-02
Copper	2.8E-05	1.0E-03	8E-04	3E-02
Lead	1.9E-05	4.0E-03	3E-02	7E+00
Manganese	3.8E-04	3.2E-03	2E-03	2E-02
Mercury	3.7E-07	1.0E-04	1E-03	3E-01
Selenium	3.9E-07	1.7E-06	1E-04	6E-04
Thallium	1.3E-05	5.6E-05	2E-01	8E-01
Zinc	7.8E-05	7.0E-01	4E-04	3E+00
ORGANICS				
Aldrin	7.6E-09	5.2E-08	3E-04	2E-03
gamma-BHC	5.0E-09	3.4E-08	2E-05	1E-04
2-Butanone	4.6E-08	1.0E-04	9E-07	2E-03
Benzoic Acid	1.3E-06	3.9E-05	3E-07	1E-05
Carbon Tetrachloride	5.7E-09	1.9E-08	8E-06	3E-05
Chlordane	2.6E-08	4.7E-06	4E-04	8E-02
Chloroform	4.6E-09	4.6E-08	5E-07	5E-06
DDT	1.1E-07	8.0E-04	2E-04	2E+00
1,4-Dichlorobenzene	4.8E-07	2.2E-05	5E-06	2E-04
Dieldrin	2.1E-08	6.4E-07	4E-04	1E-02
Ethylbenzene	4.6E-07	2.8E-04	5E-06	3E-03
Heptachlor	2.9E-08	2.0E-07	6E-05	4E-04
Heptachlor Epoxide	3.2E-09	1.0E-07	2E-04	8E-03
Methylene Chloride	5.1E-08	1.1E-05	9E-07	2E-04
PAHs- Noncarcinogenic	2.3E-06	1.4E-03	6E-06	3E-03
Pentachlorophenol	3.2E-07	1.2E-06	1E-05	4E-05
Tetrachloroethylene	8.0E-08	4.0E-04	8E-06	4E-02
Toluene	2.2E-07	1.1E-03	7E-07	4E-03
1,1,1-Trichloroethane	1.4E-06	1.7E-05	2E-05	2E-04
Trichloroethylene	4.0E-07	4.6E-05	5E-05	6E-03
Xylenes	1.1E-06	2.3E-03	5E-07	1E-03
HAZARD INDEX			2E-01	1E+01

TABLE 5-15
EXPOSURES AND RISKS ASSOCIATED WITH DIRECT CONTACT
TO SURFACE SOILS BY FUTURE ON-SITE RESIDENTS (CHILDREN)
WDI SITE

A. POTENTIAL CARCINOGENS

Chemical	CDI (mg/kg/day)		Upperbound Excess Lifetime Cancer Risks	
	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case
INORGANICS				
Arsenic	3.6E-06	1.2E-03	7E-06	2E-03
ORGANICS				
Aldrin	4.1E-09	5.9E-08	7E-08	1E-06
Benzene	1.6E-07	1.2E-04	5E-09	3E-06
BHC (delta & gamma isomers)	8.6E-08	2.5E-06	2E-07	5E-06
Carbon Tetrachloride	2.8E-09	1.3E-08	4E-10	2E-09
Chlordane	1.4E-08	5.2E-06	2E-08	7E-06
Chloroform	2.3E-09	3.2E-08	1E-11	2E-10
DDT	5.1E-08	9.0E-04	2E-08	3E-04
Dieldrin	1.1E-08	7.1E-07	2E-07	1E-05
1,4-Dichlorobenzene	2.4E-07	1.5E-05	6E-09	4E-07
Heptachlor	1.5E-08	5.5E-08	7E-08	2E-07
Heptachlor Epoxide	1.7E-09	1.2E-07	2E-08	1E-06
Methylene Chloride	2.5E-08	7.6E-06	2E-10	6E-08
PAHs - Carcinogenic	2.3E-07	3.4E-05	3E-06	4E-04
PCBs	6.8E-07	2.0E-05	5E-06	2E-04
Pentachlorophenol	2.0E-07	1.6E-06	3E-09	3E-08
Tetrachloroethylene	4.0E-08	2.7E-04	2E-09	1E-05
Trichloroethylene	2.0E-07	3.2E-05	2E-09	3E-07
Vinyl Chloride	4.4E-08	1.1E-05	1E-07	2E-05
TOTAL			2E-05	3E-03

TABLE 5-15 (cont'd)

EXPOSURES AND RISKS ASSOCIATED WITH DIRECT CONTACT
TO SURFACE SOILS BY FUTURE ON-SITE RESIDENTS (CHILDREN)

B. NONCARCINOGENS

Chemical	CDI (mg/kg/day)		CDI:RfD Ratios	
	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case
INORGANICS				
Arsenic	4.6E-05	1.4E-02	5E-02	1E+01
Antimony	4.6E-05	1.3E-03	1E-01	3E+00
Cadmium	8.8E-06	9.7E-04	9E-03	1E+00
Chromium	2.3E-04	7.9E-03	5E-02	2E+00
Copper	2.6E-04	3.8E-02	7E-03	1E+00
Lead	1.8E-04	1.5E-01	3E-01	2E+02
Manganese	3.5E-03	1.2E-01	2E-02	6E-01
Mercury	2.3E-06	8.6E-04	8E-03	3E+00
Selenium	3.6E-06	6.4E-05	1E-03	2E-02
Thallium	1.2E-04	2.1E-03	2E+00	3E+01
Zinc	7.3E-04	2.6E+01	3E-03	1E+02
ORGANICS				
Aldrin	5.1E-08	7.3E-07	2E-03	2E-02
gamma-BHC	3.3E-08	4.8E-07	1E-04	2E-03
2-Butanone	2.8E-07	8.7E-04	6E-06	2E-02
Benzoic Acid	6.9E-06	1.9E-04	2E-06	5E-05
Carbon Tetrachloride	3.5E-08	1.6E-07	5E-05	2E-04
Chlordane	1.7E-07	6.6E-05	3E-03	1E+00
Chloroform	2.8E-08	4.0E-07	3E-06	4E-05
DDT	6.4E-07	1.1E-02	1E-03	2E+01
1,4-Dichlorobenzene	3.0E-06	1.9E-04	3E-05	2E-03
Dieldrin	1.4E-07	8.9E-06	3E-03	2E-01
Ethylbenzene	2.8E-06	2.4E-03	3E-05	2E-02
Heptachlor	1.9E-07	2.8E-06	4E-04	6E-03
Heptachlor Epoxide	2.1E-08	1.5E-06	2E-03	1E-01
Methylene Chloride	3.2E-07	9.5E-05	5E-06	2E-03
PAHs- Noncarcinogenic	1.3E-05	1.2E-02	3E-05	3E-02
Pentachlorophenol	2.5E-06	2.0E-05	8E-05	7E-04
Tetrachloroethylene	4.9E-07	3.4E-03	5E-05	3E-01
Toluene	1.4E-06	9.5E-03	5E-06	3E-02
1,1,1-Trichloroethane	8.8E-06	1.4E-04	1E-04	2E-03
Trichloroethylene	2.5E-06	4.0E-04	3E-04	5E-02
Xylenes	6.7E-06	2.0E-02	3E-06	1E-02
HAZARD INDEX			2E+00	5E+02

TABLE 5-16
ASSUMPTIONS USED TO EVALUATE THE INGESTION
OF CONTAMINATED GROUNDWATER PATHWAY
UNDER FUTURE-USE CONDITIONS
WDI SITE

Parameter	Average Case	Plausible Maximum Case
On-site Residents		
Frequency of Exposure	365 days/year	365 days/year
Exposure Period	9 years	30 years
Age during Exposure Period	Adult	Adult
Average Weight over Exposure Period[a]	70 kg	70 kg
Drinking Water Consumption[b]	2 L/day	2 L/day
On-site Child Residents		
Frequency of Exposure	365 days/year	365 days/year
Exposure Period	2 years	4 years
Age during Exposure Period	0-2 years	0-4 years
Average Weight over Exposure Period[a]	10 kg	10 kg
Drinking Water Consumption	1 L/day	1 L/day
General		
Ingestion Absorption Factor	1.0	1.0
Lifetime	75 years	75 years

$$ITK_g = (C_w) \times (W) \times (G) \qquad \text{(Equation 5-7)}$$

where,

ITK_g = chemical intake from groundwater (mg/day),
C_w = chemical concentration in groundwater (mg/L),
W = daily water consumption (L/day), and
G = drinking water ingestion absorption factor.

The total intake was then converted to a chronic daily intake (CDI) as follows:

$$CDI_g = [(ITK_g)(D)(F)]/[(BW)(E)(365)] \qquad \text{(Equation 5-8)}$$

where,

CDI_g = average daily intake via groundwater (mg/kg/day);
ITK_g = daily chemical intake (mg/day);
D = duration of exposure (years);
F = frequency of exposure (days/year);
BW = body weight (kg);
E = extrapolation factor (years):
for noncarcinogens = exposure period,
for carcinogens = 75 year lifetime; and
365 = conversion factor (days/year).

Since individuals may be exposed to volatile groundwater contaminants while using water for showering or dishwashing, the risk estimates for volatile chemicals were multiplied by a factor of 2 as per EPA (1989d). Chronic daily intakes and risk estimates associated with residential drinking water consumption are presented in Table 5-17. Upperbound cancer risk estimates for ingestion of groundwater underlying the WDI site were 4×10^{-5} for average case conditions and 3×10^{-4} for plausible maximum case conditions due to arsenic. The hazard index for the plausible maximum case exceeded one.

TABLE 5-17
EXPOSURES AND RISKS ASSOCIATED WITH DOMESTIC USE OF
GROUNDWATER BY FUTURE ON-SITE RESIDENTS (ADULTS)

WDI SITE

A. POTENTIAL CARCINOGENS

Chemical	CDI (mg/kg/day)		Upperbound Excess Lifetime Cancer Risks	
	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case
INORGANICS				
Arsenic	2.0E-05	1.4E-04	4E-05	3E-04
ORGANICS				
Chloroform	9.6E-06	1.0E-04	1E-07 (*)	1E-06 (*)
Tetrachloroethylene	8.9E-06	1.3E-04	9E-07 (*)	1E-05 (*)
Trichloroethylene	9.3E-06	2.1E-04	2E-07 (*)	5E-06 (*)
TOTAL			4E-05	3E-04

B. NONCARCINOGENS

Chemical	CDI (mg/kg/day)		CDI:RfD Ratio	
	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case
INORGANICS				
Arsenic	1.7E-04	3.4E-04	2E-01	3E-01
Lead	1.1E-04	4.6E-04	2E-01	8E-01
Manganese	1.4E-02	1.7E-01	7E-02	8E-01
Mercury	4.3E-06	5.7E-05	1E-02	2E-01
ORGANICS				
Chloroform	8.0E-05	2.6E-04	2E-02 (*)	5E-02 (*)
Tetrachloroethylene	7.4E-05	3.1E-04	1E-02 (*)	6E-02 (*)
Toluene	7.4E-05	1.4E-04	5E-04 (*)	1E-03 (*)
Trichloroethylene	7.7E-05	5.1E-04	2E-02 (*)	1E-01 (*)
HAZARD INDEX			5E-01	2E+00

(*) A factor of 2 has been used to derive these risk values to account for intakes from volatilization.

Risk estimates were also quantified for a young child exposed to groundwater for the first two (2) and four (4) years of life. The child is assumed to weigh 10 kg and consume 1 L liter of water per day (EPA, 1985r). Since adult carcinogenic risk estimates are based on exposure over a lifetime (Crump et al., 1976), childhood exposure to carcinogens is included in adult cancer risk values presented above. Therefore, only noncarcinogenic endpoints will be evaluated for children. Children are expected to be more sensitive to noncarcinogens due to their low body weight.

Chronic daily intakes and risk estimates for young children consuming groundwater are presented in Table 5-18. The Hazard Index exceeded one under both average and plausible maximum conditions. Under average case conditions, no individual CDI:RfD ratio exceeded one; under plausible maximum conditions, the CDI:RfD ratio equaled or exceeded one for arsenic, lead, and manganese.

5.2.2.3 Inhalation of Subsurface Gas in Indoor Air

Individuals residing on the WDI site may be exposed to subsurface gas contaminants present in indoor air as a result of infiltration through the house's foundation as discussed in Section 4.3. Individuals are assumed to reside on site for 9 and 30 years under average and plausible maximum conditions, respectively. Exposure assumptions for this scenario are found in Table 5-19. Intake estimates for this scenario are calculated using Equations 5-5 and 5-6 in Section 5.2.1.2. Chronic daily intake and risk estimates are presented in Table 5-20. Cancer risk estimates for inhalation of volatile contaminants in indoor air ranged from 6×10^{-5} under average conditions to 6×10^{-4} under plausible maximum conditions. The CDI:RfD ratio under both average and plausible maximum conditions was less than one.

Noncarcinogenic risk estimates for inhalation of subsurface gas were also calculated for a 10 kg child residing on-site for two (2) and four (4) years under average and plausible maximum conditions. As discussed earlier, adult carcinogenic risk estimates are based on exposure over an individual's lifetime (Crump, et al., 1976); therefore, childhood exposure to carcinogens

TABLE 5-18
EXPOSURES AND RISKS ASSOCIATED WITH THE INGESTION OF
GROUNDWATER BY FUTURE ON-SITE RESIDENTS (CHILDREN)

WDI SITE

Chemical	CDI (mg/kg/day)		CDI:RfD Ratio	
	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case
INORGANICS				
Arsenic	5.9E-04	1.2E-03	6E-01	1E+00
Lead	4.0E-04	1.6E-03	7E-01	3E+00
Manganese	5.0E-02	5.8E-01	2E-01	3E+00
Mercury	1.5E-05	2.0E-04	5E-02	7E-01
ORGANICS				
Chloroform	2.8E-04	9.0E-04	6E-02 (*)	2E-01 (*)
Tetrachloroethylene	2.6E-04	1.1E-03	5E-02 (*)	2E-01 (*)
Toluene	2.6E-04	5.0E-04	2E-03 (*)	3E-03 (*)
Trichloroethylene	2.7E-04	1.8E-03	7E-02 (*)	5E-01 (*)
HAZARD INDEX			2E+00	8E+00

(*) A factor of 2 has been used to derive these risk values to account for intakes from volatilization.

TABLE 5-19
ASSUMPTIONS USED TO EVALUATE THE INHALATION PATHWAY
UNDER FUTURE-USE CONDITIONS
WDI SITE

Parameter	Average Case	Plausible Maximum Case
On-site Adult Residents		
Frequency of Detection	365 days/year	365 days/year
Duration of Exposure	24 hours/day	24 hours/day
Exposure Period	9 years	30 years
Age during Exposure Period	Adult	Adult
Average Weight over Exposure Period[a]	70 kg	70 kg
Inhalation Rate[a]	20 m^3/day	20 m^3/day
On-site Child Residents		
Frequency of Detection	365 days/year	365 days/year
Duration of Exposure	24 hours/day	24 hours/day
Exposure Period	2 years	4 years
Age during Exposure Period	0-2 years	0-4 years
Average Weight over Exposure Period[a]	10 kg	10 kg
Inhalation Rate[a]	5 m^3/day	5 m^3/day
General		
Lifetime	75 years	75 years
Inhalation Absorption Factor	1	1

[a] EPA, 1988a.

TABLE 5-20

EXPOSURE AND RISK ASSOCIATED WITH INHALATION OF VOLATILES
ORIGINATING IN SUBSURFACE GAS BY FUTURE RESIDENTS (ADULTS)

WDI SITE

A. POTENTIAL CARCINOGENS

Chemical	CDI (mg/kg/day)		Upperbound Excess Lifetime Cancer Risks	
	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case
Benzene	4.1E-05	7.2E-04	1E-06	2E-05
Carbon Tetrachloride	7.0E-07	1.3E-05	9E-08	2E-06
Chloroform	1.6E-06	6.2E-05	1E-07	5E-06
1,2-Dibromoethane	6.6E-05	2.9E-04	5E-05	2E-04
1,2-Dichloroethane	1.7E-05	1.6E-04	2E-06	1E-05
Tetrachloroethylene	3.6E-05	1.2E-04	1E-07	4E-07
Trichloroethylene	6.2E-05	3.0E-04	8E-07	4E-06
Vinyl Chloride	1.6E-05	1.3E-03	5E-06	4E-04
TOTAL			6E-05	6E-04

B. NONCARCINOGENS

Chemical	CDI (mg/kg/day)		CDI:RfD Ratios	
	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case
1,1,1-Trichloroethane	1.5E-04	4.5E-04	5E-04	1E-03
HAZARD INDEX			5E-04	1E-03

is included in the adult cancer risk values. A respiration rate of 5 m^3/day was assumed. Exposure assumptions are listed in Table 5-19. Chronic daily intakes were calculated using Equations 5-5 and 5-6.

Chronic daily intakes and risk estimates for a young child inhaling subsurface gas are presented in Table 5-21. The Hazard Index for both average and plausible maximum cases is less than one.

5.2.3 MULTI-PATHWAY EXPOSURES

Current or future populations on or near the WDI Site could be exposed to site-related contaminants through one or several exposure routes. For example, a future resident at the WDI site could ingest groundwater from an on-site well, inhale volatiles from subsurface gas, and contact contaminants in soil while gardening. The total risk to the individual will depend on the number of pathways contributing to exposure, the risk contributed by each pathway, and the toxicity of each chemical by various exposure routes (e.g., inhalation, ingestion). Since chemicals entering the body by different exposure routes may have different sites of action resulting in different toxic effects, a large uncertainty is involved in summing risks across different pathways.

5.2.3.1 Cancer Burden

In order to assess the overall impacts of the WDI site on the population, the overall cancer burden resulting from exposure to site contaminants over a 75-year lifetime is assessed. Cancer burden is quantified by summing the cancer risks for all exposure routes and then multiplying this value by the population. As presented in Section 2.2.2, currently approximately 70,000 people live within a 2-mile radius of the WDI site. Nine (9) percent of this population is under 4 years of age (6,300 individuals). The overall cancer burden for a 75 year period will be calculated for both current and future-use conditions.

TABLE 5-21

EXPOSURE AND RISK ASSOCIATED WITH INHALATION OF NONCARCINOGENIC VOLATILES ORIGINATING IN SUBSURFACE GAS BY FUTURE RESIDENTS (CHILDREN)

WDI SITE

Chemical	CDI (mg/kg/day)		CDI:RfD Ratios	
	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case
1,1,1-Trichloroethane	2.7E-04	7.8E-04	9E-04	3E-03
HAZARD INDEX			9E-04	3E-03

Under current-use conditions, the cancer burden will be calculated for off-site residents living within a 1 km radius of WDI and for students. The cancer burden for residential exposures for a 1 km radius were calculated by summing the cancer risks for 0.1 km, 0.5 km, and 1 km and then multiplying by a population of 70,000. This may result in an overestimation of the cancer burden since the population used includes areas outside a 1 km (0.6 mile) radius. Since demographic data are not available for each wind direction, no adjustment for wind direction will be made. As presented in Table 5-22, the total excess cancer risk for current residents is 4×10^{-6} and 1×10^{-5} under average and plausible maximum cases, respectively. The resulting cancer burdens are 0.3 cancers and 0.7 cancers for average and plausible maximum conditions.

For students, the total excess cancer risk is the sum of the inhalation of particulates, the inhalation of volatiles, and the trespasser scenario. The total excess cancer risk is 7×10^{-7} under average conditions and 3×10^{-5} under plausible maximum conditions. The overall cancer burden, based on a student population of 1,220, is 0.0009 cancers under average conditions and 0.04 cancers under maximum conditions.

Under future-use conditions, it was assumed that the WDI site was developed as residential property. In order to identify the potentially exposed population, it is assumed that the 43 acre (1,873,080 ft^2) site is subdivided into 7500 ft^2 lots (the minimum for residential lots in Santa Fe Springs), resulting in 250 residences. Using standard EPA (EPA, 1988h) demographic assumptions of 3.8 individuals per home, this results in a potential future exposed population of 950 individuals. The overall adult excess cancer risk is 1×10^{-4} for the average case and 1×10^{-3} under plausible maximum conditions. The potential cancer burden for future on-site residents was calculated to be 0.1 cancers under average conditions and 2 cancers under plausible maximum conditions.

TABLE 5-22

OVERALL CANCER BURDEN
OVER A 75-YEAR PERIOD
WASTE DISPOSAL, INC. SITE

Scenario	Total Excess Cancer Risk: Average Case	Total Excess Cancer Risk: Plausible Maximum Case	Total Exposed Population	Cancer Burden (# of cancers predicted in exposed population): Average Case	Cancer Burden (# of cancers predicted in exposed population): Plausible Maximum Case
Current					
Residents					
0.1 km	3E-06	1E-05			
0.5 km	6E-07	3E-06			
1 km	2E-07	1E-06			
Total Resident	4E-06	1E-05	70,000	0.3	0.7
Students	7E-07	3E-05	1,220	0.0009	0.04
Future					
Residents	1E-04	2E-03	950	0.10	2

5.3 UNCERTAINTIES IN THE RISK ASSESSMENT FOR HUMAN HEALTH

The procedures and inputs used to assess potential human health risks in this evaluation are subject to a number of uncertainties. In general, there are five main sources of uncertainty:

- -- environmental chemistry sampling and analysis,
- -- environmental parameter measurements,
- -- fate and transport modeling,
- -- toxicological data and dose-response extrapolations, and
- -- errors through combinations of the above.

These sources of uncertainty as they pertain to this assessment are summarized in Table 5-23. Primary sources of uncertainty in the WDI EA are discussed below.

Environmental chemistry sampling and analysis error can stem from the error inherent in the procedures, from a failure to take an adequate number of samples to arrive at sufficient areal resolution, from mistakes on the part of the sampler, or from the heterogeneity of the matrix being sampled. Screening techniques are not designed to be as accurate and/or precise as laboratory techniques. One of the most effective ways of minimizing procedural or systematic error is to subject the data to a strict quality control review, which has been done for this study. Even with all of the data quality rigorously assured, however, there is still error inherent in all analytical procedures. Although data for this assessment were included in a data validation program, not all data points were validated. This may lead to the inclusion of chemicals that are due to laboratory contamination rather than site conditions and may also result in the use of chemical concentration data which may be outside CLP guidelines.

The method with which the average values are calculated may represent the greatest source of uncertainty in this assessment. In using geometric means, it is assumed that the sample results are distributed log-normally. If

TABLE 5-23

ASSUMPTIONS IN THE RISK ASSESSMENT FOR HUMAN HEALTH
AND EFFECT ON RISK ESTIMATES
WDI SITE

Assumption	Effect on Risk: May Over-Estimate Risk	Effect on Risk: May Under-Estimate Risk	Effect on Risk: May Over or Under Estimate Risk
Environmental Sampling and Analysis			
Use of positively detected soil samples to characterize overall site conditions.	Moderate		
Sufficient samples may not have been taken to characterize the matrices being evaluated.			Low
Systematic or random errors in the chemical analyses may yield erroneous data.			Low
The public health evaluation is based on the chemicals of concern only, which represent a subset of the chemicals present at the site.		Low	
Use of Geometric Means as representative of average conditions.		Moderate	
High PAH detection limits in soils may result in non-detection of PAHs.		Moderate	
Exposure Parameter Estimation			
The standard assumptions regarding body weight, period exposed, life expectancy, population characteristics and lifestyle may not be representative for an actual exposure situation.			Moderate

TABLE 5-23
(CONTINUED)
ASSUMPTIONS IN THE RISK ASSESSMENT FOR HUMAN HEALTH
AND EFFECT ON RISK ESTIMATES
WDI SITE

Assumption	Effect on Risk: May Over-Estimate Risk	Effect on Risk: May Under-Estimate Risk	Effect on Risk: May Over or Under Estimate Risk
The amount of media intake is assumed to be constant over time and representative of the exposed population.		Moderate	
Concentration of contaminants remains constant over exposure period.	Moderate		
Contaminants do not degrade over time.			Moderate
Use of soil absorption factors for benzoic acid and pentachlorophenol based on uptake from liquid.	Moderate		
All intake of the chemicals of concern is assumed to come from the medium being evaluated. This does not take into account other contaminant sources such as diet or occupational exposure, exposures occurring at locations other than the exposure point being evaluated, or other environmental media which may contributed to the intake of the chemical.		Moderate	
<u>Environmental Parameter Measurement</u>			
Dermal absorption of inorganic chemicals from soil is negligible.		Low	

TABLE 5-23
(CONTINUED)
ASSUMPTIONS IN THE RISK ASSESSMENT FOR HUMAN HEALTH
AND EFFECT ON RISK ESTIMATES
WDI SITE

Assumption	Effect on Risk: May Over-Estimate Risk	Effect on Risk: May Under-Estimate Risk	Effect on Risk: May Over or Under Estimate Risk
Use of a Gaussian dispersion model to estimate air concentrations.			Moderate
Assumption that subsurface gas contaminants would infiltrate into a home at the same rate as radon.			Moderate
Toxicological Data			
Chemicals of concern in airborne particulates may be less bioavailable and toxic by inhalation than in studies from which dose-response was taken.	Moderate		
All carcinogenic PAHs are assumed to be as potent as benzo(a)pyrene.			Moderate
Chromium evaluated in this assessment was assumed to be in the hexavalent form.	High		
The cancer potencies used are upper 95 percent confidence limits derived from the linerarized multistage model. This is considered to be unlikely to underestimate the true risk.	High		
Risks are assumed to be additive. Risks may not be additive because of synergistic or antagonistic actions of other chemicals.			Moderate

TABLE 5-23
(CONTINUED)
ASSUMPTIONS IN THE RISK ASSESSMENT FOR HUMAN HEALTH
AND EFFECT ON RISK ESTIMATES
WDI SITE

Assumption	Effect on Risk: May Over-Estimate Risk	Effect on Risk: May Under-Estimate Risk	Effect on Risk: May Over or Under Estimate Risk
Cancer potencies and acceptable intake levels are primarily derived from laboratory animal studies and, when available, human epidemiological or clinical studies. Extrapolation of data from high to low doses, from one species to another, and from one exposure route to another may introduce uncertainty. In general, conservative assumptions tend to be used.	Moderate		
Assumes absorption in the lung is 100% for inorganic chemicals other than arsenic. Data concerning absorption of other inorganics in the lung are not available.	Moderate		
Toxicity values are based on lifetime exposures. The greatest length of exposure evaluated in this assessment was 30 years.			Low

the sample results are actually distributed normally (e.g., Gaussian), then the risk estimates may underestimate actual risks since arithmetic means tend to exceed geometric means. Table 5-24 presents arithmetic and geometric means for selected chemicals of concern for the WDI site.

The use of only positive detects (i.e., those samples in which a chemical was detected) for calculation of geometric means may overestimate risks for chemicals which were detected in only a small number of samples. However, it is also possible that use of positive detects only may underestimate risks, if the analytical method failed to detect the presence of the chemical due to high detection limits.

Analysis for PAHs in soils may have high analytical detection limits, due to the presence of organic matter in soil. Since only positive detects were considered for soil samples, this may result in an under-estimation of risk from PAHs or the lack of consideration of all potentially carcinogenic PAHs. However, data are not available to evaluate this fully.

Environmental parameter measurements that characterize the various media of concern at a site primarily contribute to uncertainty because of their absence. Lack of site-specific measurements dictates that estimates must be made based on literature values, extrapolations, and/or best professional judgement. In this assessment for the WDI site, some of the parameters used to estimate emission rates of particulates were based on default values (Cowherd et al., 1985). Since several of these values are raised to a power greater than or less than one in the dust generation equations shown in Appendix B, they can have a strong impact on the final outcome. Site-specific values for soil particle size were used in the models; however, estimation of airborne respirable particulates from soil particle size may have led to over- or under-estimation of risks.

There are inherent uncertainties in determining exposure parameters. For example, there are uncertainties in estimating the likelihood of an individual contacting contaminants at the site, the frequency of contact, and

TABLE 5-24

COMPARISON OF STATISTICAL METHODS FOR EVALUATION
OF ENVIRONMENTAL SAMPLING DATA

WDI SITE

CHEMICAL	ARITHMETIC MEAN	GEOMETRIC MEAN	EFFECT ON RISK VALUES (a)
SURFACE SOIL	(ug/kg)	(ug/kg)	
ARSENIC	11,000	8,000	1.38
BENZENE	260	260	1.00
CHROMIUM	24,000	23,000	1.04
LEAD	110,000	43,000	2.56
PAHs--Carcinogenic	1,100	890	1.24
PAHs--Noncarcinogenic	8,400	4,420	1.90
PCBs	3,200	2,070	1.55
SOILS--0 TO 20 FEET	(ug/kg)	(ug/kg)	
ARSENIC	11,000	6,500	1.69
BENZENE	2,700	110	24.55
CHROMIUM	29,000	26,000	1.12
LEAD	117,000	20,000	5.85
PAHs--Carcinogenic	2,450	1,340	1.83
PAHs--Noncarcinogenic	33,000	3,200	10.31
PCBs	2,540	1,130	2.25
VINYL CHLORIDE	573	31	18.48
GROUNDWATER	(ug/L)	(ug/L)	
ARSENIC	6.4	5.9	1.08
CHROMIUM	15.7	12	1.31
LEAD	5.5	4	1.38
SUBSURFACE GAS	(ug/m3)	(ug/m3)	
BENZENE	1,287	99	13.00
ETHYLENE DIBROMIDE	207	160	1.29
TETRACHLOROETHYLENE	142	88	1.61
VINYL CHLORIDE	792	38	20.84

(a) All risk values would be increased by the factor in this column.

the period of time over which such exposures would occur. All exposure estimates made in this assessment are based on reasonable assumptions about human activity patterns in the site area, not on direct observations of the behavior of specific individuals or populations. Exposure is expected to vary widely among individuals.

In general, absence of environmental parameter measurements contributes to uncertainty. Estimates must therefore be made based on literature values, regression equations, extrapolations, mathematical models and best professional judgment. In this assessment it is assumed that chemicals are absorbed from soils as readily as from other media such as water or food. It is possible that the chemicals are actually bound in a soil matrix and may not be as readily bioavailable as from food sources. Conversely, it is assumed that metals are not absorbed through the skin to a significant degree. The dermal absorption factors for benzoic acid and pentachlorophenol are based on absorption from a liquid matrix as opposed to a soil matrix. Since chemicals are generally more likely to be absorbed from a liquid matrix than a solid matrix, this assumption may result in an overestimation of dermal absorption for benzoic acid and pentachlorophenol. Under certain conditions, dermal absorption of inorganic contaminants may not be negligible (e.g., if metals are dissolved in a lipophilic solvent).

The assumption of constant concentrations in groundwater and soil over a lifetime exposure period may lead to over- or under-estimation of risks for some contaminants. Risks may be overestimated if degradation or dilution processes lead to decreasing future concentrations in the media under consideration. Risks may be underestimated if leaching from site soils leads to increased downgradient concentrations in the future. Risks may also be underestimated if a contaminant were to degrade to a more toxic contaminant (e.g., trichloroethylene to vinyl chloride).

Another source of uncertainty is the empirical equations and models used to estimate concentrations of airborne particulates and subsurface gas contaminants downwind from the WDI site. These models generally assume

steady-state conditions, such as constant contaminant concentration, constant wind speed, and average organic soil content. Also, assumptions are made regarding the physical and chemical properties of the contaminants which may not actually occur (i.e., all the chemicals found in surface soil will be bound to soil and remain so during fugitive dust generation).

Toxicological data is also a large source of uncertainty in this risk assessment. The results of animal studies are often used to predict the potential health effects of a chemical in humans. Extrapolation of toxicological data from animal tests is one of the largest sources of uncertainty in risk assessment. There may be important, but unidentified, differences in uptake, metabolism, and distribution of chemicals in the body between the test species and man. Typically, animals are administered high doses of a chemical in a standard diet. Humans, on the other hand, may be exposed to much lower doses in a highly variable diet. In these studies, animals, usually laboratory rodents, are exposed daily to the chemical agent for various periods of time up to their 2-year lifetime. Humans have a 75-year lifetime and may be exposed either intermittently or regularly for an exposure period ranging from months to a full lifetime. Even if studies in humans are available, uncertainties can be large because the diet, activity patterns, exposure duration and frequency, and individual susceptibility may not be the same in the study populations as in the individuals exposed to environmental concentrations. Since toxicity values are based on lifetime exposure for most contaminants, it may be inappropriate to use these values to estimate risks to children, who are exposed only a short fraction of a lifetime and may have a higher overall dose due to their low body weights over the exposure period.

During the evaluation of the chemical data, assumptions are made regarding the physical and chemical nature of the contaminants. In the case of chromium, all chromium is assumed to be present in the hexavalent oxidation state (CrVI). Since hexavalent chromium is more toxic than trivalent chromium (CrIII) and less prominent in the environment, this may result in an overestimation of human health risk. For polycyclic aromatic hydrocarbons

(PAHs), all carcinogenic PAHs were summed and assumed to be present as benzo(a)pyrene, one of the most potent carcinogenic PAHs. Benzo(a)pyrene accounts for 27% of the carcinogenic PAHs detected in surface soil and 19% in soils 0 to 20 feet deep; this may result in overestimation of carcinogenic PAH risk.

Cancer potency factors used in this assessment are upper-bound estimates of risk. Actual risks are not likely to be higher than these estimates, but could be considerably lower. This is an important factor contributing to the conservative nature of the risk assessment procedures used in this report. In addition, the inhalation cancer potencies for arsenic and chromium (VI) are based on epidemiologic studies of individuals exposed in occupational settings. Data are not currently available to determine if these potency factors provide reasonable estimates of cancer risks associated with exposure under the conditions considered in this risk assessment.

Uncertainties from different sources may be compounded in the risk assessment. For example, if a CDI for a contaminant measured in the environment is compared to an RfD to determine potential health hazard, the uncertainties in the concentration measurements, exposure assumptions, and the toxicities will all be present in the final risk estimate.

In order to ensure that human health is adequately protected, the endangerment assessment incorporates conservative (unlikely to underestimate risk) approaches and uncertainty factors. Therefore, the actual risks posed by a site under the scenarios considered is unlikely to be larger, but may be lower than those predicted in the assessment. This type of conservative approach was used in this risk assessment.

5.4 ECOLOGICAL ASSESSMENT

In addition to the human population, flora and fauna at and around the WDI site may also be at risk. WDI is found in the California chaparral province which extends from the coast to the mountains of southern California

(Bailey, 1980). This area contains a variety of habitats ranging from dense evergreen communities to sagebrush and grassland communities in the interior valleys and coastal plains to riparian forests containing broadleaf species along streams. The area around the WDI site is developed which limits the diversity of vegetation and animal populations that can be expected to be found.

Since the WDI site is vegetated, it may provide a habitat for animals living in the area. Gophers, for example, are known to inhabit the site. Other animals which might live in and around the WDI site may include opossum, raccoon, stripped skunk, western harvest mouse, deer mouse, California vole, and possibly blacktail jackrabbit, longtail weasel, spotted skunk, coyote, and desert cottontail (Burt and Grossenheidor, 1976).

The most commonly found birds in the summer are the wrentit, common bushtit, and rufous-sided towhee. The species found in the fall and winter are Audubon's warblers, rubycrowned kinglets, white- and golden sparrows, several types of fox sparrows, and hermit thrushes (Bailey, 1980). Other birds that area expected to be found in the area include kestrel, mourning dove, screech owl, common crow, mockingbird, and robin (Robbins et all, 1966). Other birds that might be found in the area, though it is not as likely as the previously mentioned birds due to the urbanized nature of the area, include red-tailed hawk, Swainson's hawk, western kingbird, hummingbirds, and swallows (Robbins et al., 1966).

There are a number of Federal and California endangered species that are known to inhabit Los Angeles County and may be found in the area of the WDI site. Species found on the list include: the island night lizard (Klauberina riversiana), unarmored threespine stickleback (Gasterosteus aculeatus williamsoni), slender-horned spineflower (Centrostegia leptoceras), El Segundo blue butterfly (Euphilotes (Shijimiaeoides) battoides allyni), Palos Verdes blue butterfly (Glaucopsyche lygdamus palosverdesensis), American peregrine falcon (Falco peregrinus anatum), California condor (Gymnogyps californianus), and Least Bell's vireo (Vireo bellii pusillus). The California condor has

been removed from the wild, although attempts are being made to reintroduce young condors into the wild.

There are a number of direct and indirect pathways by which wildlife can be exposed to the chemicals of potential concern at the WDI site. Direct pathways would be direct contact or ingestion of contaminated media such as soil or air. Indirect pathways, for the purposes of this assessment are those in which an animal consumes other, previously contaminated organisms. There are no surface water bodies at or near the WDI site and therefore, exposure via this medium is not evaluated in this assessment.

5.4.1 SOIL

Direct contact of contaminated soil could frequently occur among burrowing animals. Gophers build their dens below the ground surface. Skunks dig and root in the soil while searching for insects and grubs. Herbivorous animals such as gophers and rabbits may inadvertently ingest soil while eating grasses and plants. Gophers and rabbits may also be exposed to contaminated soil while grooming.

5.4.2 INGESTION OF CONTAMINATED PLANTS OR OTHER ANIMALS

The potential exists for contaminants to biomagnify via the food web. The inorganics as well as the pesticides can all be expected to bioconcentrate to some extent. Exposure to predators dependent on this habitat is of concern since they may be exposed to contaminated biota.

The most important characteristic of the pesticides is their ability to partition into lipids such as fatty animal tissue and bioaccumulate to concentrations thousands of times greater than the concentration in the environment. This biomagnification will have the greatest adverse impact on predatory animals such as birds of prey, the peregrine falcon or hawks, for example, who are at the top of the food chain. There are numerous studies relating concentrations of pesticides such as DDT to effects such as reduced

eggshell thickness and an overall decline in reproductive capacity and survival.

Gophers and rabbits are both herbivores and could be exposed by ingesting contaminated vegetation such as small land plants found at the WDI site. A large portion of the diet of most smaller birds consists of insects such as grasshoppers and crickets. Raccoons and predator birds, who might live in the area, are known to prey on small rabbits and rodents.

5.4.3 QUALITATIVE ANALYSIS OF HAZARD TO WILDLIFE

Although a quantitative assessment of environmental effects was beyond the scope of this report, certain qualitative conclusions can be drawn. The effects of inorganic contaminants on wildlife are not well documented. Wildlife may be exposed to WDI contaminants through contact with soil or ingestion of contaminated food. The adverse impact of pesticides on birds of prey and other wildlife has been demonstrated. Thus, a present or future threat to the wildlife at the WDI site may exist due to the presence of pesticides in soils and the potential for biomagnification through the food chain.

6.0 SUMMARY AND CONCLUSIONS

This Endangerment Assessment was conducted to determine the potential public health and environmental risks associated with environmental contamination at the Waste Disposal, Inc. site. An Endangerment Assessment is performed to evaluate the ramifications of the no-action remedial alternative and to provide a basis for a feasibility study to set clean-up goals.

The Waste Disposal, Inc. (WDI) site, located in Santa Fe Springs, California, received petroleum and other industrial wastes throughout its operation from 1928 until 1964. The remedial investigation at the WDI site revealed the presence in environmental media of chemicals of potential concern for effects on human health, including benzene, vinyl chloride, polycyclic aromatic hydrocarbons, organochlorine pesticides, and halogenated organic solvents. These chemicals were detected in samples collected from soil, subsurface gas, and groundwater. Arsenic, benzene, and vinyl chloride have been classified by EPA as human carcinogens following ingestion or inhalation exposure. Other chemicals found in environmental media at the WDI site have been identified by EPA as potential human carcinogens by either oral or inhalation routes. Chemicals with noncarcinogenic endpoints were also selected as chemicals of concern due to their ability to cause systemic toxicity (e.g., lead) under certain exposure conditions.

The results of the risk evaluation in Section 5 indicate that under current conditions, direct contact with surface soils by trespassers and inhalation of airborne particulates by off-site residents pose the greatest public health risk. If the land use conditions at the WDI site were to change to residential use, direct contact with surface soils by on-site residents and inhalation of subsurface gas contaminants in indoor air would pose the greatest public health risks. Table 6-1 summarizes the total upperbound lifetime excess cancer risks and the Hazard Indices for noncarcinogenic toxic effects associated with the scenarios evaluated in this assessment. The excess cancer risks and Hazard Indices represent risks which could potentially be produced by the observed concentrations. These values are not adjusted to

Table 6-1
Summary of Potential Health Risks
Waste Disposal, Inc. Site

	Total Upperbound Lifetime Excess Cancer Risks		Noncarcinogenic Hazard Index (CDI:RfD)	
	Average Case	Plausible Maximum Case	Average Case	Plausible Maximum Case
Current Use				
1. Exposure of Trespassers to surface soils	5×10^{-7}	3×10^{-5}	<1	>1 (3)
2. Exposure of off-site residents to Airborne Particulates				
- 0.1 km	3×10^{-6}	8×10^{-6}	<1	<1
- 0.5 km	5×10^{-7}	2×10^{-6}	<1	<1
- 1 km	2×10^{-7}	8×10^{-7}	<1	<1
3. Exposure of Students to Airborne Particulates	2×10^{-7}	4×10^{-7}	<1	<1
4. Exposure of off-site Residents to Airborne Volatile Chemicals				
- 0.1 km	3×10^{-7}	5×10^{-6}	<1	<1
- 0.5 km	5×10^{-8}	1×10^{-6}	<1	<1
- 1 km	2×10^{-8}	5×10^{-7}	<1	<1
5. Exposure of Students to Airborne Volatile Chemicals	3×10^{-8}	3×10^{-7}	<1	<1
Future Use				
1. Exposure of On-site Residents to Surface Soils				
- Adults	3×10^{-6}	7×10^{-4}	<1	>1 (10)
- Children (1-6 years)	2×10^{-5}	3×10^{-3}	>1 (2)	>1 (500)
2. Exposure of On-site Residents to Groundwater				
- Adults	4×10^{-5}	3×10^{-4}	<1	>1 (2)
- Children			>1 (2)	>1 (8)
3. Exposure of On-site Residents to Volatiles in Indoor Air				
- Adults	6×10^{-5}	6×10^{-4}	<1	<1
- Children			<1	<1

eliminate contributions to risk from background levels of the chemicals of concern or to incorporate other sources of chemical intake such as occupational exposure.

Under current-use conditions, the total potential excess cancer risks exceeded 10^{-6} for the trespasser direct contact with surface soils exposure scenario, and the inhalation of airborne particulates and/or vapors by residents 0.1 km from the site under plausible maximum conditions. The 10^{-6} risk level was also exceeded for residents at a 0.5 km distance in the plausible maximum case. The majority of the lifetime excess cancer risk in these scenarios is due to arsenic, PCBs, and carcinogenic PAHs. It should be noted that the surface soil concentrations of arsenic did not exceed background concentrations. The overall cancer burden over a 75 year period for current residents within 1 km of the WDI site was 0.3 under average conditions and 0.7 under plausible maximum conditions. For students, the overall cancer burden over a 75 year period was 0.0009 for the average case and 0.04 for the plausible maximum case. Under current-use conditions, only the Hazard Index for the plausible maximum case in the direct contact exposure scenario exceeded one, due primarily to lead.

Under potential future-use conditions, all exposure scenarios in the plausible maximum case had total potential upperbound cancer risks greater than or equal to 1×10^{-4}; the highest risk was associated with direct contact with soil by children aged one to six years. In the average case, upperbound cancer risks ranged from 9×10^{-6} for direct contact with soil (0-20 feet) by adults to 4×10^{-5} for direct contact with contaminated soil by children aged one to six years and consumption of drinking water. The presence of arsenic, PCBs, and carcinogenic PAHs in soil is driving the direct contact risk. Again, arsenic concentrations in soils did not exceed background concentrations. 1,2-Dibromoethane and vinyl chloride in subsurface gas are the key contributors to the inhalation risks. The overall cancer burden for a potential future residential population of 950 for a 75 year lifetime was 0.1 cancers under average conditions and 2 cancers under plausible maximum conditions. The noncarcinogenic Hazard Index was greater than or equal to one

for the direct contact and groundwater scenarios under plausible maximum conditions for all age groups, due primarily to the presence of arsenic and lead in surface soils and groundwater. Arsenic concentrations measured in downgradient wells did not exceed upgradient concentrations. Under average case conditions, the direct contact scenario for children aged one to six years had a Hazard Index greater than one. This is due primarily to the presence of lead in surface soils and groundwater.

A considerable amount of uncertainty is associated with the assessment of risks posed by a particular site. In making exposure assumptions and selecting modeling parameters, an attempt was made to use assumptions that are unlikely to underestimate risk.

7.0 REFERENCES

AGENCY FOR TOXIC SUBSTANCE AND DISEASE REGISTRY (ATSDR). 1987a. Draft Toxicological Profile for Benzene. U.S. Public Health Service.

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987b. Draft Toxicological Profile for Heptachlor/Heptachlor Epoxide. U.S. Public Health Service.

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987c. Draft Toxicological Profile for Benzo[a]pyrene. October 1987.

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987d. Draft Toxicological Profile for Selected PCBs. U.S. Public Health Service.

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987e. Draft Toxicological Profile for Cadmium. U.S. Public Health Service.

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987f. Draft Toxicological Profile for Chloroform.

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987g. Toxicological Profile for Chromium. Oak Ridge National Laboratory, October, 1987.

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1988a. Draft Toxicological Profile for Trichloroethylene. U.S. Public Health Service.

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1988b. Draft Toxicological Profile for Lead. U.S. Public Health Service.

ALBRO, P. and THOMAS, R. 1974. Intestinal absorption of hexachlorobenzene and hexachlorocyclohexane isomers in rats. Bull. Environ. Contam. Toxicol. 12: 289-294.

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio.

AMIR, D. 1973. The sites of the spermicidal action of ethylene dibromide in bulls. J. Reprod. Fert., 35:519-525.

ASTRAND, I. 1975. Uptake of Solvents in the blood and tissues of man. Scand. J. Work Environ. Health 1:199-208 (As cited in EPA, 1987) (1,4-DCB).

BAES, C.F., III, SHARP, R.D., SJOREEN, A.L., and SHOR, R.W. 1984. A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides through Agriculture. Prepared for the U.S. Department of Energy. September 1984. ORNL-5786.

BAILEY, R.G. 1980. Description of the Ecoregions of the United States. United States Department of Agriculture, Forest Service. Miscellaneous Publication Number 1391. Ogden, Utah.

BAILEY, G.W., et al. 1968. Soil Sci Soc. of Amer. Proc., 32:222-34. (As cited in HSDB, 1989).

BALYAEVA, A.P. 1967. The effects of antimony on reproduction. Gig. Truda Prof. Zabol. 11:32.

BANK, W.J. 1980. Thallium. In Spencer, P.S., and Schaumberg, H.H., eds. Experimental and Clinical Neurotoxicology. Williams and Wilkins, Baltimore. p. 571.

BANN, J.M., DeCINCO, T.J., EARL, N.W., and SUN, Y.P. 1956. The fate of aldrin and dieldrin in the animal body. Jour. Agri. Food Chem. 4:397.

BARDODEJ, Z. and BARDODEJOVA, E. 1970. Biotransformation of ethylbenzene, styrene, and alpha-methylstyrene in man. Am. Ind. Hyg. Assoc. J. 31:206-209.

BARKER, J.G., PATRICK, G.C., and MAJOR, D. 1987. Natural attenuation of aromatic hydrocarbons in a shallow sand aquifer. Winter 1987. Ground Water Monitoring Rev. p. 64-71.

BARNES, D., et al. 1987. Reference dose (RfD): description and use in health risk assessment. Appendix A in IRIS Supportive Documentation Volume 1. Office of Health and Environmental Assessment, U.S. EPA, Washington, D.C. EPA 600/8-861032a.

BARTLETT, R.J. and KIMBLE, J.M. 1976. Behavior of chromium in soils: 1. Trivalent forms. J. Environ. Qual. 5:379-382.

BARTLETT, R.J. and JAMES, B.R. 1979. Behavior of chromium in soils: III. Oxidation. J. Environ. Qual. 8:31-25.

BIDLEMAN, T.F., BILLINGS, W.N., and FOREMAN, W.T. 1986. Vapor-particle partitioning of semivolatile organic compounds: Estimates from field collections. Environ. Sci. Technol. 10:1038.

BIRNBAUM, L. 1986. Personal Communication. National Institute for Environmental Health Sciences. April, 1986.

BODEK, I., LYMAN, W.J., REEHL, W.F., and ROSENBLATT, D.H. 1988. Environmental Inorganic Chemistry. Properties, Processes and Estimation Methods. Pergamon Press, Inc., Elmsford, New York.

BOUBLIK, T., FRIED, V., and HALA, E. 1984. The vapor pressures of pure substances: selected values of the temperature dependence of the vapor pressures of some pure substances in the normal and low-pressure region. Vol. 17. Elsevier Sci. Publ., Amsterdam, Netherlands.

BOURGEOIS, M., DOOMS-GOOSSENS, A., KNOCKAERT, D., SPRENGERS, D., VAN BOVEN, M. and VAN TITTELBOOM, T. 1986. Mercury intoxication after topical application of a metallic mercury ointment. Dermatol. 172:48-51.

BOUWER, E.J. and McCARTY, P.L. 1983. Transformations of 1- and 2- carbon halogenated aliphatic organic compounds under methanogenic conditions. Appl. Environ. Microbiol. 45(4):1286-1294.

BOWEN, H.J.M. 1979. Environmental Chemistry of the Elements. Academic Press, N.Y.

BREIVOGEL, M. 1955. Director of Planning, Regional Planning Commission, County of Los Angeles. Letter to Waste Disposal, Inc., 12731 E. Los Nietos Road, Whittier, California. Attention: Marvin Pitts, President. March 18.

BREIVOGEL, M. 1956. Director of Planning, Regional Planning Commission, County of Los Angeles. Letter to Air Pollution Control District, 5201 Santa Fe Avenue, Los Angeles, California. April 6.

BRUCKNER, J.V., MACKENZIE, W.F., MURALIDHARA, S., et al. 1986. Oral toxicity of carbon tetrachloride: acute, subacute and chronic studies in rats. Fund. Appl. Toxicol. 6(1):16-34.

BUBEN, J.A. and O'FLAHERTY, E.J. 1985. Delineation of the role of metabolism in the hepatotoxicity of trichloroethylene and perchloroethylene: A dose-effect study. Toxico. Appl. Pharmacol. 78:105-122.

BUCKHARD, L.P., ARMSTRONG, D.E., and ANDREN, A.W. 1985. Partitioning behavior of polychlorinated biphenyls. Environ. Sci. Technol. 19:590-596.

BURKHALTER, J. and BALSTER, R.L. 1979. Behavioral teratology evaluation of chloroform in mice. Neurobehav. Toxicol. 1:199-205.

BURT, W.H. and GROSSENHEIDER, R.P. 1976. A Field Guide to the Mammals. Third Edition. Houghton Mifflin Company, Boston, Massachusetts.

CABRAL, J.R.P., HALL, R.K., ROSSI, L., BRONKZYK, S.A., and SHUBIK, K.P. 1982. Effects of long-term intake of DDT on rats. Tumori. 68:11-17 (As cited in EPA, 1989a).

CACI. 1989. Demographic and income forecast report for Santa Fe Springs and Los Nietos. March, 1989.

CALIFORNIA DEPARTMENT OF HEALTH SERVICES (DHS). 1986. The Califonia Site Mitigation Decision Tree Manual. Toxic Substances Control Division, Alternative Technology and Policy Development Section. May 1986.

CALIFORNIA DEPARTMENT OF HEALTH SERVICES (DHS). 1987. The Development of Applied Action Levels for Soil Contact: A Scenario for the Exposure of Humans to Soil in a Residential Setting. Final Draft. Prepared by the Toxic Substances Control Division.

CALIFORNIA DEPARTMENT OF HEALTH SERVICES (DHS). 1989. Applied Action Levels for Human Receptors. Pentachlorophenol. Stat of California Department of Health Services, Toxic Substances Control Division, Sacramento, CA.

CAREY, A.E., GOWEN, J.A., FOREHAND, T.J., TAI, H., and WEIRSMA, G.B. 1980. Heavy metal concentrations in soils of five United States cities: 1972 urban monitoring program. Pestic. Monit. J. 13:150-154.

CASTRO, C.E. and BELSER, N.O. 1968. Biodehalogenation. Reductive dehalogenation of the biocides ethylene, ethylene dibromide, 1,2-dibromo-3-chloropropane, and 2,3-dibromobutane. In Soil. Environ. Sci. Technol. 2:770-783.

CAVENDER, F.L., CASEY, H.W., SALEM, H., SWENBERG, J.A., and GARALLA, E.J. 1983. A 90-day vapor inhalation toxicity study of methyl ethyl ketone. Fund. Appl. Toxicol. 3:264-270.

CHANEY, R.L., STERRETT, S.B., and MIELKE, H.W. 1984. The potential for heavy metal exposure from urban gardens and soils. In Proceedings Symp. on Heavy Metals in Urban Gardens, Dist. Columbia Extension Service, Washington, D.C. pp. 37-84.

CHEMICAL INDUSTRY INSTITUTE OF TOXICOLOGY (CIIT). 1980. A twenty-four month inhalation toxicology study in Fischer-344 rats exposed to atmospheric toluene. Executive Summary and Data Tables. October 15, 1980.

CHEN, C., CHUANG, Y., YOU, S., LIN, T., and WU, H. 1986. A retrospective study on malignant neoplasms of bladder, lung and liver in blackfoot disease endemic area in Taiwan. Brit. J. Cancer 53:399-405.

CHIN, B.H., McKELVEY, J.A., TYLER, T.R., CALISTI, L.J., KOZBELT, S.J., and SULLIVAN, L.J. 1980. Absorption, distribution and excretion of ethylbenzene, ethylcyclohexane and methyl ethylbenzene isomers in rats. Bull. Environ. Contam. Toxicol. 24:477-483.

CLAYTON, G.D. and CLAYTON, F.E., eds. 1984. Patty's Industrial Hygiene and Toxicology. 3rd. ed. John Wiley and Sons, New York. p. 1916.

COATES, D. E. 1959. County of Los Angeles, Department of County Engineer, Industrial Waste Division, Dump Inspection Report, Whittier Waste Disposal, 12731 Los Nietos Road. June 29.

COLLINS, L. J. 1959. County of Los Angeles, Department of County Engineer, Industrial Waste Division, Dump Inspection Report, Waste Disposal, Inc., 12731 Los Nietos Road, Santa Fe Springs. April 1.

CONNOR, J.J., SHACKLETTE, H.T., et al. 1975. Background Geochemistry of Some Rocks, Soils, Plants and Vegetables in the Conterminous United States. Geological Survey Professional Paper 574-F. U.S. Government Printing Office, Washington, D.C.

COOVER, M.P. and SIMS, R.C. 1987. The effect of temperature on polycyclic aromatic hydrocarbon persistence in an unacclimated agricultural soil. Haz. Waste Haz. Mat. 4:69-82.

COTTON, F.A. and WILKINSON, G. 1982. Advanced Inorganic Chemistry. 3rd ed. John Wiley and Sons, New York.

COULSON, E.J., REMINGTON, R.E., and LYNCH, K.M. 1935. Metabolism in the rat of the naturally occuring arsenic of shrimp as compared with arsenic trioxide. J. Nutr. 10:255-276.

COUNCIL FOR AGRICULTURAL SCIENCE AND TECHNOLOGY (CAST). 1976. Farm Chem., 138(13), 36(1075); Rep. No. 59, September 15, 1976. (As cited in Deichmann, 1981.)

COWHERD, C., Jr., et al. 1985. Rapid Assessment of Exposure to Particulate Emissions from Surface Contamination Sites. Office of Health and Environmental Assessment, U.S. EPA, Washington, D.C. 600/8-85/002.

COX, D.P. 1975. Methylation of Arsenic. In: Gould, R.F., (ed.), Arsenical Pesticides, ACS Symposium Series.

CRECELIUS, E.A., JOHNSON, C.J., and HOFER, G.C. 1974. Contamination of soils near a copper smelter by arsenic, antimony and lead. Water Air Soil Pollut., 3:337.

CRECELIUS, E.A., BOTHNER, M.H. and CARPENTER, R. 1975. Geochemistries of arsenic, antimony, and related elements in sediments of Puget Sound. Environ. Sci. Technol. 9(4):325-333.

CRONKITE, E.P., DREW, R.T., INOUE, T., and BULLIS, J.E. 1985. Benzene hematotoxicity and leukemogenesis. Am. J. Ind. Med. p. 133.

CRUMP, K., HOEL, D., LANGLEY, C., and PETO, R. 1976. Fundamental Carcinogenic Processes and their implications to low dose risk assessment. Cancer Research 36:2973-2979.

DALLAS, C.E., WEIR, F.W., FELDMAN, S., PUTCHA, L., and BRUCKNER, J.V. 1983. The uptake and distribution of 1,1-dichloroethylene in rats during inhalation exposure. Toxicol. Appl. Pharmacol. 68:140-151 (As cited in EPA, 1987) (1,4-DCB).

DAMES AND MOORE. 1985. Summary of Findings Phase II Investigation Waste Disposal Inc. Site for Redevelopment Agency City of Santa Fe Springs, California. March 14.

DAMES AND MOORE. 1986a. Draft Report Floor Sampling Survey Shallow Soil Vapor Survey, Toxo Spray Dust, Inc. Site, Santa Fe Springs, California. Dames and Moore Job No. 13262-017-042. August 19.

DAMES AND MOORE. 1986b. Report for Soil Sampling Program, Toxo Spray Dust, Inc. Site, Santa Fe Springs, California. Dames and Moore Job No. 13262-017-042.

DAVIS, K.J. 1965. Patholoy report on mice fed Dieldrin, Aldrin, Heptachlor, and Heptachlor Epoxide for two years. Internal FDA memorandum to Dr. A.J. Lehrman, July 19.

DAVIS, K.J. and FITZHUGH, O.G. 1962. Tumorigenic potential of aldrin and dieldrin for mice. Toxicol. Appl. Pharmacol. 4:187-189.

DEAN, R.B. 1981. Use of lognormal statistics in environmental monitoring. In: Chemicals and Water Use. Vol. I. Cooper, W.J., ed. Ann Arbor Science, Ann Arbor, MI.

DEICHMANN, W.B. 1981. Halogenated cyclic hydrocarbons. In Patty's Industrial Hygiene and Toxicology. 3rd edition. John Wiley and Sons, New York. pp.3740-3749.

DEICHMANN, W.R. 1972. Toxicology of DDT and related chlorinated hydrocarbon pesticides. J. Occup. Med. 14:285-292.

DELLA PORTA, G.B., TERRACINI, B., and SHUBIK, K.P. 1961. Induction with carbon tetrachloride of liver cell carcinomas in hamsters. J. Natl. Cancer Inst. 26:855-863.

DEPARTMENT OF WATER RESOURCES (DWR). 1961. Bulletin No. 104. Planned Utilization of the Groundwater Basins of the Coastal Plain of Los Angeles County. Appendix A. Groundwater Geology.

DESKON, H.D. 1979. Carbon Tetrachloride. In: Grayson, M., and Eckroth, D. (eds.) Kirk-Othmer Encyclopedia of Chemical Technology, 3rd ed., Vol. 5. New York: John Wiley and Sons. pp.693-703.

DOW CHEMICAL COMPANY. 1955. 60-Week Feeding Study with Dogs for Heptchlor. MRID No. 00061912 (Unpublished) (As cited in EPA, 1989a).

EBASCO SERVICES INCORPORATED (EBASCO). 1988a. Field Sampling and Analysis Plan. Waste Disposal Inc. June 1988.

EBASCO SERVICES INCORPORATED (EBASCO). 1988b. Work Plan for Remedial Investigation/Feasibility Study. Waste Disposal Inc. April 1988.

EBASCO SERVICES INCORPORATED (EBASCO). 1989a. Final Subsurface Gas Characterization Report. Waste Disposal Inc., May 1989.

EBASCO SERVICES INCORPORATED (EBASCO). 1989b. Draft Soil Characterization Report. Waste Disposal Inc., March 1989.

EBASCO SERVICES INCORPORATED (EBASCO). 1989c. Groundwater Characterization Report. Waste Disposal Inc. May 1989.

EBASCO SERVICES INCORPORATED (EBASCO). 1989d. Letter to Mike Raybourn from Dan Melchior. April 26, 1989.

EBASCO SERVICES INCORPORATED (EBASCO). 1989e. Databases with Environmental Sampling Results. June 1989.

EBASCO SERVICES INCORPORATED (EBASCO). 1989f. Personal Communication with Dan Melchior, Site Manager. November 7, 1989.

EDWARDS, K., JACKSON, N., and JONES, A.R. 1970. Studies with alkylating esters. II. A chemical interpretation through metabolic studies of the antifertility effects of ethylene dimethanesulphonate and ethylene dibromide. Biochem. Pharmacol., 19:1783-1789.

EDWARDS, J., HESTON, W.E., and DALTON, H.A. 1942. Induction of carbon tetrachloride hepatoma in strain L mice. J. Natl. Cancer Inst. 3:297-301.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1978. Occupational Health Guidelines for Chlordane, Lindane, Malathion, and Warfarin. Chemical, Physical and Biological Properties of Compounds Present at Hazardous Waste Sites. Vols. I, II, and III. Office of Waste Programs Enforcement. Office of Solid Waste and Emergency Response. September, 1978.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. December. EPA 440/4-79-029a and b. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980a. Ambient Water Quality Criteria Document for DDT. Office of Water Regulations and Standards, Washington, D.C. EPA 440/5-80-038.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980b. Ambient Water Quality Criteria for Antimony. Office of Water Regulations and Standards, Washington, D.C. EPA 440/5-80-020.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980c. Ambient Water Quality Criteria for Chromium. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-80-035.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984a. Health Effects Assessment for Lindane. Office of Emergency and Remedial Response. Washington, D.C. September 1984. EPA 540/1-86-056.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984b. Health Assessment Document for Carbon Tetrachloride. Environmental Criteria and Assessment Office. Cincinnati, Ohio. September. EPA-600/8-82-001F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984c. Health Effects Assessment for Chloroform. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-010

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984d. Health Effects Assessment Document for DDT. Office of Emergency and Remedial Response, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984e. Health Effects Assessment for Pentachlorophenol. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-043

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984f. Health Effects Assessment for Polychlorinated Biphenyls. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-004.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984g. Health Effects Assessment for Polycyclic Aromatic Hydrocarbons (PAHs). Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-013.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984h. Health Effects Assessment for Benzo[a]pyrene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-022.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984i. Health Effects Assessment for 1,1,1-Trichloroethane. Final Draft. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-H005.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984j. Health Effects Assessment for Vinyl Chloride. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-036.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984k. Health Effects Assessment for Xylene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. EPA 540/1-86-006.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984l. Health Assessment Document for Inorganic Arsenic. Office of Health and Environmental Assessment, Washington, D.C. March 1984. EPA 600/8-83-021F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984m. Health Effects Assessment for Arsenic. Environmental Criteria and Assessment Office. Cincinnati, Ohio. September 1984. EPA 540/1-86-037.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984n. Health Assessment Document for Chromium. Environmental Criteria and Assessment Office, Research Triangle Park, North Carolina. EPA 600/8-83-014F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984o. Health Effects Assessment for Lead. Environmental Criteria and Assessment Office, Cincinnati, Ohio. EPA/540/1-86-055.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984p. Health Assessment Document for Manganese. Environmental Criteria and Assessment Office, Environmental Protection Agency, Cincinnati, Ohio. Final Report. August 1984. EPA-600/8-83-013F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984q. Health Effects Assessment for Mercury. Environmental Criteria and Assessment Office. Cincinnati, Ohio. EPA 540/1-86-042.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984r. Health Effects Assessment for Selenium. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-058.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984s. Health Effects Assessment for 1,2-Dichloroethane. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-002. PB86-134137.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984t. Health Effects Assessment for Tetrachloroethylene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-009. PB86-134202.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984u. Health Effects Assessment for Toluene. Environmental Criteria and Assessment Office. Cincinnati, Ohio. September 1984. EPA 540/1-86-033. PB86-134442.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984v. Health Effects Assessment for Methyl Ethyl ketone. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-003. PB86-134145.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984x. Health Effects Assessment for Zinc (and Compounds). Office of Emergency and Remedial Response. September. Washington, D.C. EPA 540/1-86-048.

ENVIRONMENAL PROTECTION AGENCY (EPA). 1984w. Health Effects Assessment of Ethylbenzene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-008.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Drinking Water Criteria Document for Benzene. Final Draft. April 1985. Office of Drinking Water, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. Drinking Water Criteria Document for Carbon Tetrachloride. Final Draft. January. Office of Drinking Water. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985c. Health Assessment Document for Chloroform. Environmental Criteria and Assessment Office, Research Triangle Park, North Carolina. September 1985. EPA 600/8-84-004F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985d. Health Assessment Document for 1,2-Dichloroethane. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA 600/8-84-006F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985e. Drinking Water Criteria Document for Heptachlor, Heptachlor Epoxide, and Chlordane. Final Draft. March, 1985. PB86-117991 EPA 600/x.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985f. Drinking Water Criteria Document for Pentachlorophenol. Final Draft. Office of Drinking Water. March 1985. EPA 600X-84-177-1

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985g. Health Effects Criteria Document on Polychlorinated Biphenyls. Final Draft. Office of Drinking Water, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985h. Health Assessment Document for Tetrachloroethylene (Perchloroethylene). Office of Health and Environmental Assessment, Washington, D.C. July 1985. EPA 600/8-82-005F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985i. Drinking Water Criteria Document for Tetrachloroethylene. Office of Drinking Water, Criteria and Standards Division. April 1985. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985j. Drinking Water Criteria Document for Toluene. Final Draft. Office of Drinking Water, Washington, D.C. March 1985.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985k. Drinking Water Criteria for 1,1,1-Trichloroethane. Draft. Office of Drinking Water, Criteria and Standards Division. Washington, D.C. January, 1985.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 19851. Health Assessment Document for Trichloroethylene. Environmental Criteria and Assessment Office. Research Triangle Park, N.C. EPA 600/8-82/006F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985m. Drinking Water Criteria Document on Vinyl Chloride. Final Draft. Office of Drinking Water. January 1985. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985n. Drinking Water Criteria Document for Cadmium. Final Draft. Office of Drinking Water, Washington, D.C. April 1985. PB86-117934.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985o. Updated Mutagenicity and Carcinogenicity Assessment of Cadmium. Addendum to the Health Assessment Document for Cadmium (May 1981; EPA/600/8-81/023). Office of Health and Environmental Assessment, June 1985. Washington, D.C. EPA 600/8-83-025F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985p. Drinking Water Criteria Document for Copper. Final Draft. Environmental Criteria and Assessment Office, Cincinnati, Ohio. March 1985. EPA-600/X-84-190-1. NTIS publ. No. PB86-118239.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985q. Draft Health Advisory for Lead. Office of Drinking Water. September 30, 1985. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985r. Development of Statistical Distribution or Ranges of Standard Factors Used in Exposure Assessments. Office of Health and Environmental Assessment. OHEA-5-161. March 1985.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985s. Health Assessment Document for Dichloromethane. Office of Health and Environmental Assessment. February. Washington, D.C. EPA/600/8-82004F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985t. Drinking Water Criteria Document for *ortho*-Dichlorobenzene, *meta*-Dichlorobenzene, and *para*-Dichlorobenzene (Draft). Office of Drinking Water, Washington, D.C. June 1985. PB86-117918 (1,4-DCB).

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985u. Drinking Water Criteria Document for Lindane. Final Draft. Environmental Criteria and Assessment Office. March 1984. ECAO-CIN-402.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985v. Addendum to the Health Assessment Document for Dichloromethane, Office of Health and Environmental Assessment, Washington, D.C. September 1985. EPA/600/8-82-004F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986a. Superfund Public Health Evaluation Manual. Office of Emergency and Remedial Response. EPA/540/1-86/060.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986b. Guidelines for Carcinogenic risk assessment. Fed. Reg. 51:33992-24003. September 24.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986c. Guidelines for Health Risk Assessment of Chemical Mixtures. Federal Register. Vol. 51. (185):341014-34025. September 24.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986d. Guidelines for Noncarcinogenic Risk Assessment. Federal Register. Vol. 51. September 24.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986e. Health and Environmental Effects Profile for Naphthalene. Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986f. Draft Guidelines for Classifying Ground Water. Office of Groundwater Protection. December 1986.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986g. Verified Reference Doses (RfDs) of the U.S. EPA. ECAO-CIN-475

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987a. Health Advisory for Ethylbenzene. Office of Drinking Water. March 1987. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987b. Health Advisory. Heptachlor and Heptachlor Epoxide. Office of Drinking Water. March 31, 1987. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987c. Health Advisory for Toluene. Office of Drinking Water. March 1987. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987d. Draft Health Advisory for Trichloroethylene. Office of Drinking Water. March 31, 1987. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987e. Addendum to the Health Assessment Document for Trichloroethylene: Update Carcinogenicity Assessment for Trichloroethylene. Review draft. June 1987. EPA 600/8-82/006FA.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987f. Health Advisory for Xylenes. Office of Drinking Water. March 1987. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987g. Drinking Water Criteria Document for Copper. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria & Assessment Office.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987h. Draft Health Advisory for Mercury. Office of Drinking Water. September 30. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987i. Superfund Program: Interim Guidance on Compliance with Applicable or Relevant and Appropriate Requirements. August 27, 1987. Fed. Reg. 52:32496-32499.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987j. Health Advisory for *ortho-*, *meta-*, and *para-*Dichlorobenzene. Office of Drinking Water, Washington, D.C. March 31, 1987 (1,4-DCB).

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988a. Integrated Risk Information System (IRIS). November. Environmental Criteria and Assessment Office, Cincinnati, OH.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988b. Updated Health Effects Assessment for tetrachloroethylene. Prepared by the Office of Health and Environmental Assessment. Environmental Criteria and Assessment Office. Cincinnati, Ohio. Office of Emergency and Remedial Response, Washington, D.C. ECAO-CIN-N009a.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988c. Updated Health Effects Assessment for Trichloroethylenes. Prepared by the Office of Health and Environmental Assessment. Environmental Criteria and Assessment Office. Cincinnati, Ohio. Office of Emergency and Remedial Response. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988d. Special Report on Ingested Inorganic Arsenic; Skin Cancer Nutritional Essentiality. Risk Assessment Forum. U.S. EPA. Washington, D.C. EPA/625/3-87/013f.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988e. Superfund Exposure Assessment Manual. Office of Emergency and Remedial Response. April 1988.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988f. Exposure Factors Handbook. Office of Health and Environmental Assessment. Washington, D.C. September 1988.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988g. Updated Health Effects Document for Thallium and compounds. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, Ohio. for the Office of Emergency and Remedial Response, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988h. Hazard Ranking System (HRS) for Uncontrolled Hazardous Substance Releases; Appendix A of the National Oil and Hazardous Substances Contingency Plan. Proposed Rule. 40 CFR Part 300. December 23, 1988.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989b. June Quarterly Update for HEA and HEED Chemicals. Memorandum from Chris DeRosa. July 15, 1988. Environmental Criteria and Assessment Office. Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989c. Memorandum from Winston Porter. Interim Final Guidance for Soil Ingestion Rates. Office of Solid Waste and Emergency Response. January 1989. OSWER Directive 9850.4

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989d. Health Effects Assessment Summary Tables. Prepared by the Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. April 1989 (1,4-DCB).

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989e. Memorandum from John Kemmerer. EPA's Comments on Draft Endangerment Assessment for Waste Disposal, Inc., Sept. 19, 1989.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989f. Interim guidance on establishing soil lead cleanup levels at Superfund Sites. Memorandum from Henry L. Longest II and Bruce Diamond. Office of Solid Waste and Emergency Response. OSWER Directive #9355.4-02. September 7, 1989.

EPSTEIN, S.S. 1976. Carcinogenicity of heptachlor and chlordane. Sci. Total Environ. 6:103-154.

FAO/WHO JOINT EXPERT COMMITTEE ON FOOD ADDITIVES. 1974. Toxicological Evaluation of Some Food Additives Including Anticaking Agents, Antimicrobials, Antioxidants, Emulsifiers, and Thickening Agents. FAO Nutr. Rep. Ser. No. 53A, Rome. WHO/Food Add./74.5. (As cited in Opdyke, 1979).

FARMER, W.J., YANG, M.S., LETEY, J., SPENCER, W.F., and ROULIER, M.H. 1978. Land disposal of hexachlorobenzene wastes: controlling vapor movement in soils. San Antonio, Texas. 4th Annual Symposium on Land Disposal.

FEDERAL REGISTER. 1988. Part 5, Environmental Protection Agency. August 18, 1988. pp. 31516-31578.

FEDERAL REGISTER. 1989. Part II, Environmental Protection Agency. May 22, 1989. pp. 22062-22160.

FELDMAN, R.J., and MAIBACH, H.I. 1970. Absorption of some organic compounds through the skin in man. J. Invest. Dermatol. Applied Pharmacol., 28:126-132.

FELDMAN, R.J., and MAIBACH, H.I. 1974. Percutaneous penetration of some pesticides and herbicides in man. Toxicol. Appl. Pharmacol. 28:126-132.

FERON, V.J., HENDRIKSEN, C.F.M., SPEEK, A.J., TIL, H.P., and SPIT, B.J. 1981. Lifespan oral toxicity study of vinyl chloride in rats. Food Cosmet. Toxicol. 19:317-333.

FITZHUGH, O., NELSON, A., LAUG, E., and KUNZE, F. 1950. Chronic oral toxicities of mercuric-phenyl and mercuric salts. Arch. Ind. Hyg. Occup. Med. 2:433-441.

FITZHUGH, O., NELSON, A.A., and QUAIFE, M.L. 1964. Chronic oral toxicity of aldrin and dieldrin in rats and dogs. Food Cosmet. Toxicol. 2:551-562.

FOREMAN, W.T., and BIDLEMAN, T.F. 1985. Vapor pressure estimates of industrial PCBs and commercial fluids using gas chromatography retention data. J. Chromatogr. 330:203-216.

FROST, R.R. and GRIFFIN, R.A. 1977. Effect of pH on adsorption of copper, zinc, and cadmium from landfill leachate by clay minerals. J. Environ. Sci. Health 12:138-156.

FUKUDA, K., TAKEMOTO, K., and TSURUTA, H. 1983. Inhalation carcinogenicity of trichloroethylene in mice and rats. Ind. Health. 21:243-254.

FULLER, W.H. 1978. Investigation of Landfill Leachate Pollutant Attenuation by Soils. Prepared for the Municipal Environmental Research Laboratory, Office of Research and Development. U.S. Environmental Protection Agency, Cincinnati, Ohio. EPA 600/12-78-158, NTIS PB. 286-995. August, 1978.

GARDINER, J. 1974. The chemistry of cadmium in natural waters - II. The adsorption of cadmium on river muds and naturally occurring solids. Water Research. 8:157-164.

GIBSON, D.T., KOCH, J.R., and KALLIO, R.E. 1966. Oxidative degradation of aromatic hydrocarbons by microorganisms. Enzymatic formation of catechol from benzene. Biochemistry; 7(7):2653-2662.

GIBSON, D.T., KOCH, J.R., and KALLIO, R.E. 1968. Oxidative degradation of aromatic hydrocarbons by microorganisms: I. Enzymatic formation of catechol from benzene. Biochemistry: 653-2662.

GIBSON, J.E., and BECKER, B.A. 1970. Placental transfer, embryo toxicity, and teratogenicity of thallium sulfate in normal and potassium-deficient rats. Toxicol. Appl. Pharmacol. 16:120-132.

GLOVER, J.R. 1967. Selenium in human urine: A tentative maximum allowable concentration for industrial and rural populations. Ann. Occup. Hyg. 10:3-10.

GOSSELIN, R.E., SMITH, R.P., and HODGE, H.C., eds. 1984. Clinical Toxicology of Commercial Products. 5th ed. Williams and Wilkins, Baltimore, Maryland. P. 203

GRANCICH, J.B. 1958. County of Los Angeles, Department of County Engineer, Industrial Waste Division, Dump Inspection Report, Waste Disposal Inc., 12751 Los Nietos Road. October 23.

GRIFFIN, R.A. and CHIAN, E.S.K. 1980. Attenuation of water soluble polychlorinated biphenyls by earth materials. Solid and Hazardous Waste Research Division. Municipal Environmental Research Laboratory. Cincinnati, Ohio.

GUY, R.D. and CHAKRABARTI, C.L. 1976. Studies of metal-organic interactions in model systems pertaining to natural waters. Can. J. Chem. 54:2600-2611.

HAMMOND, P.B., and BELILES, R.P. 1980. Metals. In: Casarett and Doull's Toxicology: The Basic Science of Poisons, 2nd Ed., Doull, J., Klaassen, C.D., and Amdur, M.O. (eds.), Macmillan Publishing Co., New York, Toronto, and London. pp. 421-428.

HANNINEN, H., ESKELININ, L., HUSMAN, K., and NURMINEEN, M. 1976. Behavioral effects of long-term exposure to a mixture of organic solvents. Scand. J. Work Environ. Health. (As cited in EPA, 1987c). 2:240-255.

HANSCH, C., and LEO, A. 1979. Substituent Constants for Correlation Analysis in Chemistry and Biology. John Wiley and Sons, Inc., New York.

HANSCH, C., and LEO, A.J. 1985. MedChem Project, Issue No. 16, Pomona College, Claremont, CA.

HAQUE, R. (ed.). 1970. Dynamics Exposure and Hazard Assessment of Toxic Chemicals. Ann Arbor Sci. Publ. Inc., Ann Arbor, Michigan.

HARDIN, B.D., BOND, G.P., SIKOV, M.R., ANDREW, F.D., BELILES, R.P., and NIEMEIER, R.W. 1981. Testing of selected workplace chemicals for teratogenic potential. Scand. J. Work Environ. Health 7:66-75.

HAWLEY, J.K. 1985. Assessment of health risk form exposure to contaminated soil. J. Risk Anal., 5:289-302.

HAYES, W.J., JR. 1982. Pesticides Studied in Man. Williams and Williams, Baltimore. Pp. 385-389.

HAZARDOUS SUBSTANCE DATA BASE (HSDB). 1989. Toxnet On-line Service. March, 1989.

HEATH, D.F., and VANDEKAR, M. 1964. Toxicity and metabolism of dieldrin in rats. Br. J. Ind. Med. 21:269-279.

HEYWOOD, R., SORTWELL, R.J., NOEL, P.R.B., et al. 1979. Safety evaluation of toothpaste containing chloroform. III. Long-term study in beagle dogs. J. Environ. Pathol. Toxicol. 2:835-851 (As cited in EPA, 1985c)

HUANG, C.P., ELLIOTT, H.A., and ASHMEAD, R.M. 1977. Interfacial reactions and the fate of heavy metals in soil-water systems. J. Water Pollut. Control. 49:745-756.

HUDAK, A., and UNGVARY, G. 1978. Embryotoxic effects of benzene and its methyl derivatives: toluene, xylene. Toxicology 11:55-63.

HUNTER, C.G., ROBINSON, J., and ROBERTS, M. 1969. Pharmacodynamics of dieldrin (HEOD). Ingestion by human subjects for 18-24 months and post exposure for eight months. Arch. Environ. Health 18:12-21.

HURSH, J.B., CLARKSON, T.W., MILES, E.F. and GOLDSMITH, L.A. 1989. Percutaneous Absorption of Mercury Vapor By Man. Arch. Environ. Health. 44:120-127.

HUTZINGER, S., SAFE, S., and ZITKO, V. 1974. The Chemistry of PCBs. Cleveland, OH, Chemical Rubber Co. Publishing.

INGLE, L. 1952. Arch. Ind. Hyg. Occup. Med. 6,354 (as cited in Deichmann, 1981).

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1975. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. Vol. 9: Some Aziridines, N-, S-, and O- Mustards, and Seleniums. World Health Organization. Lyon, France.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1977. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man: Some Fumigants, the Herbicides. 2,4-D and 2,4,5-T, Chlorinated Dibenzodioxins and Miscellaneous Industrial Chemicals. Vol. 15. pp. 195-209.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1979a. IARC Monographs on the Evaluation of the Carcinogenic Risks of Chemicals to Humans. Vol. 20: Some Halogenated Hydrocarbons. World Health Organization. Lyon, France. pp. 143-204.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1979b. Evaluation of the carcinogenic risk of chemicals to humans. Vinyl chloride, polyvinyl chloride and vinyl chloride. Vinyl acetate polymers. World Health Organization. Lyon, France: 19:377-401; 419-438.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1982a. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Supplement 4. Chemicals, Industrial Processes and Industries Associated with Cancer in Humans. World Health Organization, Lyon, France. pp. 205-206.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1982b. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 27: Some Aromatic Amines, Anthraquinones and Nitroso Compounds, and Inorganic Fluorides Used in Drinking-Water and Dental Preparations. World Health Organization, Lyon, France.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1983. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Vol. 32: Polynuclear Aromatic Compounds; Part 1, Chemical, Environmental and Experimental Data. World Health Organization, Lyon, France.

INTERNATIONAL RESEARCH and DEVELOPMENT CORPORATION (IRDC). 1973. Unpublished report to Velsicol Chemical Corporation. Eighteen Month Oral Carcinogenic Study in Mice, December 14, (as cited in Epstein, 1976).

ITO, N., NAGASAKI, H., ARAI, M. et al. 1973. Histological and ultrastructural studies on the hepatocarcinogenicity of benzenehexachloride in mice. J. Natl. Cancer Inst. 51:817-826.

IVANKOVIC, S., and PREUSSMAN, R. 1975. Absence of toxic and carcinogenic effects after administration of high doses of chromic oxide pigment in subacute and long-term feeding experiments in rats. Fd. Cosmet. Toxicol. 13:347-351.

JABER, H.M., MABEY, W.R., LIV, A.T., CHOU, T.W., JOHNSON, H.L., MILL, T., PODOLL, R.T., and WINTERLE, J.S. 1984. Data Acquisition for Environmental Transport and Fate Screening. Office of Health and Environmental Assessment, U.S. EPA, Washington, D.C. EPA 600/6-84-009.

JENKINS, L.J., JONES, R.A., and SIEGEL, J. 1970. Long-term inhalation screening studies of benzene, toluene, o-xylene and cumene on experimental animals. Toxicol. Appl. Pharmacol. 16:818-823.

JENNE, E.A. 1968. Controls on Mn, Fe, Co, Ni, Cu, and Zn Concentrations in Soils and Water: The significant role of hydrous Mn and Fe oxides. In: Gould, R.F., (ed.). Trace Inorganics in Water: Advances in Chemistry 73. Washington, D.C., American Chemical Society.

JORGENSON, T.A., MEIERHENRY, E.F., RUSHBROOK, C.J., et al. 1985. Carcinogenicity of chloroform in drinking water to male Osborne-Mendel rats and female B6C3F1 mice. Fund. Appl. Toxicol. (U.S.A.); 5(4): 760-769.

KABATA-PENDIAS, A. and PENDIAS, H. 1984. Trace Elements in Soils and Plants. CRC Press, Boca Raton.

KAO, J.K., PATTERSON, F.K., and HALL, J. 1985. Skin penetration and metabolism of topically applied chemicals in six mammalian species including man: An in vitro study with benzo[a]pyrene and testosterone. Toxicol. Appl. Pharmacol. 81:502-516 (As cited in ATSDR, 1987c).

KARNOFSKY, D.A., RIDGWAY, L.P., and PATTERSON, P.A. 1950. Production of achondroplasia in the chick embryo with thallium. Proc. Soc. Exp. Biol. Med. 73:255-259.

KIMBROUGH, R.D., SQUIRE, R.A., LINDER, R.E., STRANDBERG, J.D., MONTALI, R.J. and BURSE, V.W. 1975. Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. J. Natl. Cancer Inst. 55:1453.

KIMMERLE, G., and EBEN, A. 1973. Metabolism, excretion, and toxicology of trichloroethylene after inhalation. 1. Experimental exposure on rats. Arch. Toxicol. 30:115

KIMURA, E.T., EBERT, D.M., and DODGE, P.W. 1971. Acute toxicity and limits of solvent residue for sixteen organic solvents. Toxicol. Appl. Pharmacol. 19:699-704.

KINZELL, J.J. 1981. A toxicologic assessment of pentachlorophenol in lactating dairy cattle. Ph.D. dissertation. Michigan State University.

KJELLSTRAND, P., KANJE, M., MANSSON, L., BJERKAMO, M., MORTENSEN, I., LANKE, J., and HOLMQUIST, B. 1981. Trichloroethylene: Effects on body and organ weights in mice, rats, and gerbils. Toxicology; 21:105-115 (As cited in ATSDR, 1988a).

KLEMMER, H.W., WONG, L., SATO, M.M., REICHERT, E.L., KORSAK, R.J., and RASHAD, M.N. 1980. Clinical findings in workers exposed to pentachlorophenol. Arch. Environ. Contam. Toxicol. 9(6): 715-725

KOLLER, L. 1979. Methylmercury toxicity, model no. 158. In: Jones, T.C., Hackel, D.B., and Migaki, G., (eds.) Handbook: Animal Models of Human Disease. Registry of Comparative Pathology, Armed Forces Institute of Pathology, Washington, D.C.

KORTE, N.E., SKOPP, J., FULLER, W.H., NIEVLA, E.E., and ALESII, B.A. 1976. Trace element movement in soils: Influence of soil physical and chemical properties. Soil Sci. 122:350-525.

LABELLE, C.W., and BRIEGER, H. 1955. Vapor toxicity of a composite solvent and its principal components. Arch. Ind. Health 12:623-627.

LAI, J.C.K., LEUNG, T.K.C., and LIM, K. 1982. Activities of the mitochondrial and linked isocitric dehydrogenase in different regions of the rat brain. Gerontology 28:81-85.

LANDE, S.S., DURKIN, P.R., CHRISTOPHER, D.H., HOWARD, P.H., and SAXENA, J. 1976. Investigation of Selected Potential Environmental Contaminants: Ketonic Solvents. U.S. Environmental Protection Agency, Washington, D.C. EPA 560/2-76-003.

LANG, E.P., NELSON, A.A., FITZHUGH, O.G., and KUNZE, F.M. 1950. Liver Cell Alteration and DDT storage in the liver of the rat induced by dietary levels of 1-50 ppm DDT. J. Pharmacol. Exp. Ther. 98:268-273.

LAXEN, D.H., DAVISON, W. and WOOL, C. 1984. Manganese chemistry in rivers and streams. Geochem. Cosmichm. Acta. 48:2107-2111.

LEUNG, T.K.C., LAI, J.C.K., and LIM, L. 1981. The regional distribution of monoamine oxidase activities towards different substrates: Effects in rat brain of chronic administration of manganese chloride and of aging. J. Neurochem. 36:2037-2043.

LITTLE, ARTHUR D., INC. 1976. Literature study of selected potential environmental contaminants: antimony and its compounds. pp. 5-15. 251-438.

LOEKKE, H. 1984. Water, Air, Soil Pollut. 22:373-87. (As cited in HSDB, 1989).

LU, P., METCALF, R.L., and COLE, L.K. 1978. The environmental fate of ^{14}C-pentachlorophenol in laboratory model ecosystem, in: Pentachlorophenol, Environmental Science Research Series, Vol. 12, Rao, K.R., (ed.). Plenum Press, NY.

LUCIER, G.W., RUMBAUGH, R.C., McCOY, Z., HASS, R., HARVAN, D., and ALBRO, K. 1986. Ingestion of soil contaminated with 2,3,7,8-tetrachlorodibenzo-p-dixoin (TCDD) alters hepatic enzyme activities in rats. Fund. and Appl. Toxicol. 6:364-481.

LYMAN, W.L., REEHL, W.F., and ROSENBLATT, D.T. 1982. Handbook of Chemical Property Estimation Methods. McGraw-Hill Book Co., New York; pp. 15-25.

MABEY, W.R., SMITH, J.W., PODOLL, R.T., JOHNSON, H.L., MILL, T., CHOU, T.W., GATES, J., PATRIDGE, I.W., JABER, H. and VANDENBERG, D. 1982. Aquatic Fate Process Data for Organic Priority Pollutants. Prepared for SRI International. Prepared for Monitoring and Data Support Division, Office of Water Regulations and Standards. Washington, D.C.

MACKAY, D., and SHIV, W.Y. 1981. A critical review of Henry's Law Constants for Chemicals of Environmental Interest. J. Phys. Chem. Ref. Data: 19:1175-1179.

MACKAY, D. and SHIU, W.Y. 1977. Aqueous solubility of polynuclear aromatic hydrocarbons. J. Chem. and Engr. Data. 22:399-402.

MACKENZIE, R.D., BYERRUM, R.V., DECKER, C.F., HOPPERT, C.A., LONGHAM, F.L. 1958. Chronic toxicity studies II. Hexavalent and trivalent chromium administered in drinking water to rats. Arch. Ind. Health. 18:232-234.

MACKAY, D., SHIU, W.Y., BILLINGTON, J., and HUANG, G.L. 1983. Physical chemical properties of polychlorinated biphenyls in Mackay, D., et al., eds. Physical Behavior of PCB's in the Great Lakes. Ann Arbor Science, Ann Arbor, Michigan.

MAGORIAN, T.R., WOOD, K.G., MICHALOVIC, J.G., PEK, S.L. and VAN LIER, M.M. 1974. Water Pollution by Thallium and Related Metals (NTIS PB 253 333, Springfield, Virginia) (cited in EPA, 1979).

MAHAFFEY, K.R. 1978. Environmental exposure to lead. In: Nriagu, J.O. (ed.). The Biochemistry of Lead in the Environment: Part B. Biological Effects. Elsevier/North-Holland Biomedical Press, New York. 397 pages.

MALTONI, C., CONTI, B., COTTI, G., and BELPOGGI, F. 1985a. Experimental studies on benzene carcinogenicity at the Bologna Institute of Oncology: Current results and ongoing research. Am. J. Ind. Med. 7:415-446.

MALTONI, C., LEFEMINE, G., COTTI, G., CHIECO, P., and PATELLA, V. 1985b. Experimental Research on Vinylidine Chloride Carcinogenesis. In Archives of Research on Industrial Carcinogenesis. Princeton Scientific Publishers, Princeton, New Jersey. 3 vols.

MANCUSO, T.F. 1975. International Conference on Heavy Metals in the Environment. Toronto, Canada.

MAXFIELD, D.J., RODRIGUEZ, M., BUETTNER, M., DAVIS, J., FORBES, L., KOVACS, R., RUSSEL, W., SCHULTZ, L., SMITH, R., STANTON, J., and WAI, C.M. 1974. Heavy metal pollution in the sediments of the Coeur D'Alene River delta. July. Environ. Pollut. 7(1):1-6.

McCONNEL, E.E., LUCIER, G.W., RUNBAUGH, R.C., ALBRO, P.W., HARVAN, D.J., HASS, J.R., and HARRIS, M.W. 1984. Dioxin in soil: Bioavailability after ingestion by rats and guinea pigs. Science 223:1077-1079.

McDUFFIE, B., RUSSEL, D.J., and NATICHAR, J.J. 1984. Potential for migration into groundwater. Proc. Triangle Conf. on Environ. Tech. Duke University. March 6-8, 1984.

McLACHLAN, J.A., and DIXON, R.L. 1972. Gonadal function in mice exposed prenatally to p,p'-DDT. Toxicol. Appl. Pharmacol. 22:327.

MEDLEY, A. 1959. County of Los Angeles, Department of County Engineer, Industrial Waste Division, Dump Inspection Report, Whittier Waste Disposal, 12731 Los Nietos Road. August 6.

MESTITZOVA, M. 1967. On reproduction studies on the occurrence of cataracts in rats after long-term feeding of the insecticide heptachlor. Experientia 23:42-43.

MIETTINEN, J.K. 1973. Absorption and elimination of dietary mercury (Hg^{2+}) and methylmercury in man. In Miller, M., and Clarkson, T., (eds.) Mercury, Mercurials and Mercaptans. Charles C. Thomas, Springfield, IL.

MIKESELL, D. and BOYD, S.A. 1988. Enhancement of pentachlorophenol degradation in soil through induced anaerobiosis and bioaugmentation with anaerobic sewage sludge. Environ. Sci. Technol. 22(12):1411-1414.

MITSUMORI, K., MAITA, K., TORU, S., SHUJI, T. and SHIRASU, U. 1981. Carcinogenicity of methylmercury chloride in ICR mice: preliminary note on renal carcinogenesis. Cancer Letters. 12:305-310. Elsevier/North-Holland Scientific Publishers Ltd.

MOORE, B. 1958. Memorandum to T.T. Otteson, Department of County Engineer, County of Los Angeles, regarding Waste Disposal, Inc., I-629. September 10.

MOORE, B. 1962. County of Los Angeles, Department of County Engineer, Industrial Waste Division, Dump Inspection Report, Whittier Waste Disposal Inc., 12731 Los Nietos Road. March 9.

MOORE, J.W. and RAMAMOORTHY, S. 1984. Heavy Metals in Natural Waters. Springer-Verlag, New York.

MORIYA, M., OHTA, T., WATANABE, K., MIYAZAWA, T., KATO, K., and SHIRASU, Y. 1983. Further mutagenicity studies on pesticides in bacterial reversion assay systems. Mutat. Res. 116(3-4): 185-216

MUNIR, K.M., SOMAN, C.S. and SAMATI, V. 1983. Hexachlorocyclohexane-induced tumorigenicity in mice under different experimental conditions. Tumori. 69:383-386.

MURPHY, D.A. and KORSCHGEN, L.F. 1970. Reproduction growth and tissue residues of deer fed dieldrin. Wildlife Mgmt. 34:887-903.

MURPHY, T., POKOJOWCZYK, J.C., and MULLIN, M.D. 1983. In: Physical Behavior of PCBs in the Great Lakes. MacKay, D., Patterson, S., Eisenreich, S.J., and Simmons, M.S. (eds.) Ann Arbor Science. Ann Arbor, MI, pp. 49-58.

MURPHY, T.J., MULLIN, M.D., and MEYER, J.A. 1987. Equilibration of PCBs and toxaphenes with air and water. Env. Sci. Technol. 21(2): 155-162.

MURRAY, F.J., SCHWETZ, B.A., MCBRIDE, J.G., STAPLES, R.E. 1979. Toxicity of inhaled chloroform in pregnant mice and their offspring. Toxicol. Appl. Pharmacol. 50:525-522

NASH, R.G. 1983. Comparative volatilization and dissipation rates of several pesticides form soil. J. Agric. Food Chem. 31:210-7.

NASH, R.G. and WOOLSON, E.A. 1967. Science, 157:924-927. (As cited in HSDB, 1989).

NATIONAL ACADEMY OF SCIENCES (NAS). 1976a. Drinking Water and Health. vol.

NATIONAL ACADEMY OF SCIENCE (NAS). 1976b. Health Effects of Benzene: A Review Committee on Toxicology, Assembly of Life Sciences. National Research Council, Washington, D.C.

NATIONAL CANCER INSTITUTE (NCI). 1976c. Carcinogenesis Bioassay of Chloroform. CAS No. 67-66-3. NCI Carcinogenesis Technical Report Series No. 0. Bethesda, Maryland. DHEW (NIH) Publication No. 76

NATIONAL CANCER INSTITUTE (NCI). 1976d. Carcinogenesis Bioassay of Trichloroethylene. CAS No. 79-01-6. Carcinogenesis Technical Report Series No. 2. PB-264-122.

NATIONAL CANCER INSTITUTE (NCI). 1977a. Bioassay of 1,1,1-Trichloroethane for possible carcinogenicity. NCI Carcinogenesis Tech. Rep. Ser. No. 3.

NATIONAL CANCER INSTITUTE (NCI). 1977b. Bioassay of heptachlor for possible carcinogenicity. CAS. No. 76-44-8. Technical Report Series No. 9. Bethesda, MD: Carcinogenesis Program, Division of Cancer Cause and Prevention, NCI, National Institutes of Health. DHEW Publication No. 77-809.

NATIONAL CANCER INSTITUTE (NCI). 1977c. Bioassay of Tetrachloroethylene for Possible Carcinogenicity. CAS No. 127-18-4. NCI Carcinogenesis Technical Report Series No. 13. Washington, D.C. DHEW (NIH) Publication No. 77-813

NATIONAL CANCER INSTITUTE (NCI). 1977d. Bioassay of Chlordane for Possible Carcinogenicity. NCI Carcinogenesis Technical Report Series No. 8, 117p.

NATIONAL CANCER INSTITUTE (NCI). 1978a. Bioassay of Aldrin and Dieldrin for Possible Carcinogenicity. Bethesda, Maryland. National Institutes of Health. NCI-C6-TR-21. DHEW Publ. No. 78-821 184 pp.

NATIONAL CANCER INSTITUTE (NCI). 1978b. Bioassays of DDT, TDE, and p,p'-DDE for Possible Carcinogenicity. CAS No. 40-29-3, 72-54-8, 72-55-9. NCI Carcinogenesis Technical Report Series No. 131.

NATIONAL CANCER INSTITUTE (NCI). 1978d. Bioassay of 1,2-Dichloroethane for Possible Carcinogenicity. NCI Carcinogenesis Technical Report Series No.55. DHEW Publ. No. (NIH) 78-1361.

NATIONAL CANCER INSTITUTE (NCI). 1978c. Bioassay of 1,2-dibromoethane for possible carcinogenicity. NCI Carcinogenicity Tech. Rep. Ser. No. 86. PB-288-428, p. 64.

NATIONAL CANCER INSTITUTE (NCI). 1978e. Bioassay of Aroclor 1254 for Possible Carcinogenicity. CAS No. 27323-18-8. NCI Carcinogenesis Technical Report Series No. 38. DHEW (NIH) Publication No. 78-838.

NATIONAL COFFEE ASSOCIATION (NCA). 1983. Twenty-fourth-Month Oncogenicity Study of Methylene Chloride in Mice. Unpublished report prepared by Hazelton Laboratories, Inc., Vienna, Virginia.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1977. Criteria for a Recommended Standard--Occupational Exposure to Coal Tar Products. DHEW (NIOSH) 78-107.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1978a. Special Occupational Hazard Review: Aldrin/Dieldrin. US DHEW Publ. No. 78-201.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1978b. Special Occupational Hazard Review: DDT. USDHEW Publ. No. 78-200.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1985. Pocket Guide to Chemical Hazards. USDHHS.

NATIONAL OCEANIC AND ATMOSPHERIC ADMINISTRATION (NOAA). 1983. Climatic Atlas of the United States.

NATIONAL RESEARCH COUNCIL (NRC). 1982. An Assessment of Health Risks of Seven Pesticides Used for Termite Control. August 1982. NRC-P901. NTIS Publ. PB83-136374.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1982. Carcinogenesis Bioassay of 1,2-dibromoethane in F344 rats and B6C3F1 mice (inhalation study), NTP-80-28. NIH Pub. No. 82-1766.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1983. Carcinogenesis Studies of Trichloroethylene (Without Epichlorohydrin), CAS No. 79-01-6, in F344/N rats and B6C3F_1 mice (Gavage Studies). Draft. August 1983. NTP 81-84, NTP TR 243.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1984. Annual Plan for Fiscal Year 1984. Research Triangle Park, North Carolina. DHHS, Public Health Services. NTP-84-023 for 1,1,1- TCA.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1986a. Toxicology and Carcinogenesis Bioassay of Benzene in F344/N Rats and B6CF1 Mice. Gavage Studies. NTP Technical Report Series #289. Research Triangle Park, North Carolina.

NATIONAL TOXICOLOGY PROGRAM. 1986b. Toxicology and carcinogenesis studies of 1,4-dichlorobenzene in F344/N rats and B6C3F1 mice. U.S. DHHS, NIH Technical Report Series No. 319 (1,4-DCB).

NATIONAL TOXICOLOGY PROGRAM (NTP). 1986c. Toxicology and Carcinogenesis Studies of xylenes (mixed) [60% m-xylene; 14% p-xylene, 9% o-xylene; 17% ethylbenzene] in F344/N rats and B63F1 mice (Gavage studies). December 1986. NTP TR 327.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1986d. Toxicology and Carcinogenesis Studies of Tetrachloroethylene (Perchloroethylene) CAS No. 127-18-4 in F344/N Rats and B6C3F1 Mice (Inhalation Studies). NTP Technical Report Series No. 311. Research Triangle Park, North Carolina. DHEW (NIH) Publication No. 86-2567.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1988. Board Draft NTP technical report on the toxicology and carcinogenesis studies of pentachlorophenol. CAS No. 87-86-5.

NAWROT, P.S., and STAPLES, R.E. 1979. Embryo-fetal toxicity and teratogenicity of benzene and toluene in the mouse. Teratology 19:41A.

NEAL, J., and RIGDON, R.H. 1967. Gastric tumors in mice fed benzo(a)pyrene: A quantitative study. Tex. Rep. Biol. Med. 25:553-557.

NORBACK, D.H., and WELTMAN, R.H. 1985. Polychlorinated biphenyl induction of hepatocellular carcinoma in the Sprague-Dawley rat. Environ. Health Perspect. 1:134-143.

O'GRADY, M.D. 1955. Minutes of Hearing, Regional Planning Commission, County of Los Angeles, Special Permit Case 1032. Memorandum to Milton Breivogel, Director of Planning, Regional Planning Commission, County of Los Angeles. March 3.

ONG, H.L., SWANSON, V.E., and BISQUE, R.E. 1970. Natural organic acids as agents of chemical weathering. U.S. Geological Survey Professional Paper 700-C:C130-137.

OPDYKE, D.L.J. 1979. Benzoic acid. Monographs on fragrance raw materials. Food Cosmet. Toxicol. 17(Supplement):715-722

OTT, M.G., TOWNSEND, J.C., FISHBECK, W.A., and LANGNER, R.A. 1978. Mortality among individuals occupationally exposed to benzene. Arch. Environ. Health 33:3-10.

OTT, W.R. 1988. A physical explanation of the lognormality of pollution concentrations. Presented at the 81st Annual Meeting of the Air Pollution and Hazardous Waste Management Association. June 19-24, 1988. Dallas, TX.

OTTESON, T.T. 1957. Memorandum to J.L. Partin, Department of County Engineer, County of Los Angeles, regarding Waste Disposal Inc., I-629-10. May 9.

OTTOLENGHI, A.D., HASEMAN, J.K., and SUGGS, F. 1974. Teratogenic effects of aldrin, dieldrin, and endrin in hamsters and mice. Teratology 9:11-16.

PAL, D., WEBER, H.B., and OVERCASH, M.R. 1974. Fate of polychlorinated biphenyls in soil-plant systems. Residue Reviews, Vol. 74. pp. 45-98.

PARRIS, G.E., and BRINKMAN, F.E. 1976. Reactions which relate to the environmental mobility of arsenic and antimony. II. Oxidation of trimethylarsine and trimethylstibine. Environ. Sci. Technol. 10(12):1128-1134.

PARSONS, F., WOOD, P.R., and DEMARCO, J. 1984. Transformations of tetrachloroethene and trichloroethene in microcosms and groundwater. J. Am. Water Works Assoc. 76:56-59.

PIERCE, R.H. and VICTOR, D.M. 1978. The fate of pentachlorophenol in an aquatic ecosystem. In Pentachlorophenol: Chemistry, Pharmacology, and Environmental Toxicology. Plenum Press, New York. pp. 41-52.

PITTS, M. 1955. Waste Disposal, Inc. 12731 E. Los Nietos Road, Whittier, California. Request for Hearing for Special Permit 1032. January 24.

POHLMAN, A.A., and McCALL, J.G. 1986. Kinetics of metal dissolution from forest soil by soluble organic acids. J. Environ Quality 15:86-92.

POIGER, H., and SCHLATTER, C. 1980. Influence of solvents and adsorbants on dermal and intestinal absorption of TCDD. Fd. Cosmet. Toxicol., 18:477-481.

PORTER, K.G., MCMASTA, D., ELMES, M.E., and LOVE, A.G. 1977. Anemia and low serum-copper during zinc therapy. Lancet, Vol. 8:744.

POURBAIX, M. 1963. Atlas d'Equilibres Electrochemiques. Gauthier-Villar and Co., Paris.

PRASAD, A.S., SCHOOMAKER, E.B., ORTEGA, J., et al. 1975. Zinc deficiency in sickle cell disease. Clin. Chem. 21:582-587.

PRICE, J. 1985a. Santa Fe Springs Chief Engineer. Telephone conversation with Theresa Gioia (EPA) regarding source of City water and its usage. August 5.

PRICE, J. 1985b. Santa Fe Springs Chief Engineer. Telephone conversation with Theresa Gioia (EPA) regarding aquifers from which the City's groundwater is drawn. October 15.

QUAST, J.F., HUMISTON, C.G., WADE, C.E., BALLARD, J., BEYER, J.E., SCHWETZ, R.W., and NORRIS, J.M. 1983. A chronic toxicity and oncogenicity study in rats and subchronic toxicity study in dogs on ingested vinylidene chloride. Fund. Appl. Toxicol. 3: 55-62.

RAMAMOORTHY, S., and RUST, B.R. 1978. Heavy metal exchange processes in sediment-water systems. Environ. Geol. 2:165-172.

RAO, P.S.C. and DAVIDSON, J.M. 1982. Retention and transformation of selected pesticides and phosphorus in soil-water systems: A Critical Review. U.S. EPA, Athens, Georgia. EPA 600/53-82-060.

RAPPAPORT, R.A. and EISENREICH, S.J. 1984. Chromatographic determination of octanol-water partition coefficients (Kow's) for 58 polychlorinated biphenyl congeners. Env. Sci. Technol. 18:163-170.

RASHID, M.A, and LEONARD, J.D. 1973. Modifications in the solubility and precipitation behavior of various metals as a result of their interactions with sedimentary humic acid. Chem. Geol. 11: 89-97.

RAY-BETTLEY, F., and O'SHEA, J.A. 1975. The absorption of arsenic and its relation to carcinoma. Br. J. Dermatol. 96:563 (as cited in EPA, 19841).

RILEY, R.A., CHART, I.S., DOSS, A., et al. 1980. Para-dichlorobenzene: Long-term Inhalation Study in the Rat. ICI Report Nos.CTL/P/447. August 1980 (1,4-DCB).

RINSKY, R.A., YOUNG, R.J., and SMITH, A.B. 1981. Leukemia in benzene workers. Am. J. Ind. Med. 3:217-245.

ROBBINS, C.S., BRUUN, B., and ZIM, H.S. 1966. A Guide to the Identification of Birds of North America. Golden Press, Western Publishing Company, New York, New York.

ROBERTS, M.S., ANDERSON, R.A. and SWARBRICK, J. 1977. Permeability of human epidermis to phenolic compounds. J. Pharm. Pharmac. 29:677-683.

ROBERTS, R.J., CHERRY, J.A., and SCHWARTZ, F.W. 1982. A case study of a chemical spill: polychlorinated biphenyls (PCBs). Water Resources Research. Vol. 18, No. 3. pp. 525-534.

ROBSON, A.M., KISSANE, J.M., ELVICK, N.H., and PUNDAVELA, L. 1969. Pentachlorophenol poisoning in a nursery for newborn infants. I. Clinical features and treatment. J. Pediat. 75: 309-316

ROSSI, L., RAVERA, M., REPETTI, G., and SANTI., L. 1977. Long-term administration of DDT or phenobarbital-NA in Wistar rats. Int. J. Cancer, 19:179-185 (as cited in EPA, 1989a).

ROWE, V.K., SPENCER. 1952., H.C., McCOLLISTER, D.D., HOLLINGSWORTH, R.L., and ADAMS, E.M., A.M.A. Toxicity of ethylene dibromide determined on experimental animals. Arch. Ind. Hyg. Occup. Med., 6:158-173. 1952. (As cited in Clayton & Clayton, 1984).

SANDBORN, J.R., et al. 1977. The Degradation of Selected Pesticides in Soil: A Review of the Published Literature. EPA 600/9-77-022. (As cited in HSDB, 1989).

SANTODONATO, J., HOWARD, P., and BASU, D. 1981. Health and ecological assessment of polynuclear aromatic hydrocarbons. J. Envrion. Pathol. Toxicol. 5:1-364.

SAX, N.I. 1984. Dangerous Properties of Industrial Materials. 6th ed. Van Nostrand Reinhold Co., New York. p. 378.

SAXENA, J., ed. 1986. Hazard Assessment of Chemicals. Volume 5. Hemisphere Pub. Co., Washington.

SCHAEFFER, E., GREIM, H., and GOESSNER, W. 1984. Pathology of chronic polychlorinated biphenyl (PCB) feeding in rats. Toxicol. Appl. Pharmacol. 75:278-288.

SCHAUM, J.L. 1984. Risk analysis of TCDD contaminated soil. Office of Helath and Environmental Assessment, U.S. Environmental Protection Agency, Washington, D.C. EPA 600/8-84-031.

SCHELLENBERG, L., LEUENBERGER, C., and SCHWARZENBACH, R.P. 1984. Sorption of chlorinated phenols by natural sediments and aquifer materials. Environ. Sci. Technol. 18:652-65.

SCHMAHL, D. 1955. Testing of Naphthalene and Anthracene as Carcinogenic Agents in the Rat. Z. Krebsforsch. 60: 697-710 (German with English translation).

SCHMIDT, R. 1973. Effects of 1,1,1-trichloro-2,2-bis(p-chlorophenol)-ethane (DDT) on the prenatal development of the mouse. Biol. Rundsch. 11:316-317 (Ger.).

SCHROEDER, H.A., MITCHNER, M., and NASAR, A.P. 1970. Zirconium, niobium, antimony, vanadium, and lead in rats: Lifetime studies. J. Nutr. 100:59-66.

SCHWETZ, B.A., LEONG, B.K.J., and GEHRING, P.J. 1974a. Embryo- and fetotoxicity of inhaled carbon tetrachloride, 1,1-dichloroethane and methyl ethyl ketone in rats. Toxicol. Appl. Pharmacol. 28:452-464.

SCHWETZ, B.A., LEONG, B.K.J., and GEHRING, P.J. 1974b. Embryo- and fetotoxicity of inhaled chloroform in rats. Toxicol. Appl. Pharmacol. 28:442-451 (As cited in EPA, 1985c)

SCHWETZ, B.A., LEONG, B.K.J., and GEHRING, P.J. 1975. The effect of maternally inhaled trichloroethylene, perchloroethylene, methyl chloroform, and methylene chloride on embryonal and fetal development in mice and rats. Toxicol. Appl. Pharmacol. 55:207-219.

SCHWETZ, B.A., QUAST, J.F., KEELER, P.A., HUMISTON, C.G., and KOCIBA, R.J. 1978. Results of 2-year toxicity and reproduction studies on pentachlorophenol in rats. In: Rao, K.R., ed. Pentachlorophenol: Chemistry, Pharmacology and Environmental Toxicology. Plenum Press, New York. p. 301

SCOTT, A. 1983. Review of Existing Instrumentation and Evaluation for Research to Determine Levels of Radon at a Proposed Building Site. January 1983.

SHACKLETTE, H.T., and BOERNGEN, J.G. 1984. Element Concentrations in Soils and Other Surficial Materials of the Conterminous United States. U.S. Government Printing Office, Washington, D.C. U.S. Geological Survey Professional Paper 1270.

SHIU, W.Y. and MACKAY, D. 1986. A critical review of aqueous solubilities vapor pressures, Henry's constants, and octanol-water partition coefficients of the polychlorinated biphenyls. J. Phys. Chem. Ref. Data. 15:911-929.

SHORT, R.D., MINOR, J.L., WINSTON, J.M. SEIFTER, J. and LEE, C.C. 1978. Toxicol. Appl. Pharmacol., 46:173. (As cited in Clayton and Clayton, 1984).

SKOG, E. and WAHLBERG, J.E. 1964. A Comparative Investigation of the Percutaneous Absorption of Metal Compounds in the Guinea Pig by Means of the Radioactive Isotpoes: ^{51}Cr, ^{58}Co, ^{65}Zn, ^{110m}Ag, ^{115m}Cd, ^{203}Hg. J. Investigative Dermatol. 36:187-200.

SNYDER, C.A., GOLDSTEIN, B.D., SELLAKUMAR, A.R., BROMBERG, I., LASKIN, S., and ALBERT, R.E. 1980. The inhalation toxicology of benzene: Incidence of hematopoietic neoplasms and hematotoxicity in AKR/J and C57BL/6J mice. Toxicol Appl Pharm. p. 361.

STEWART, R.D., HAKE, C.L., FORSTER, H.V., LEBRUN, A.J., PETERSON, J.F., and WU, A. 1974. Tetrachloroethylene: Development of a biologic standard for the industrial worker by breath analysis. Report No. NIOSH-MCOW-ENUM-PCE-74-6, Medical College of Wisconsin, Milwaukee, Wisconsin.

TABAK, H.H., QUAVE, S.A., MASHNI, C.L., and BARTH, E.F. 1981. Biodegradability studies with organic priority pollutant compounds. J. Water Pollut. Contr. Fed.; 53:1503-1518.

TAKEUCHI, Y., ONO, Y., HISANAGA, N., et al. 1983. An experimental study of the combined effects of n-hexane and methyl ethyl ketone. Br. J. Ind. Med. 40:199-203.

TENNEKES, H.A., WRIGHT, A.S., DIX, K.M., DOEMAN, J.H. 1981. Effects of dieldrin, diet and bedding on enzyme function and tumor incidence in livers of male CF-1 mice. Cancer Res. 41:3615-3620.

TERRACINI, B., TESTA, M.C., CABRAL, J.R.P., and DAY, N. 1973. The effects of long-term feeding of DDT to BALB/c mice. Int. J. Cancer. 11:747-764.

THOMPSON, D.J., WATNET, S.D., ROBINSON, V.V. 1974. Teratology studies on orally administered chloroform in the rat and rabbit. Toxicol. Appl. Pharmacol.; 29:348-357. (As cited in ATSDR, 1987f)

THORPE, E. and WALKER, A.I.T. 1973. Toxicology of dieldrin (HEOD). II. Comparative long-term oral toxicity studies in mice with dieldrin, DDT, phenobarbitone, beta-HCH and gamma-HCH. Food Cosmet. Toxicol. 11:433-442.

THUN, M.J., SCHNORR, T.M., SMITH, A.B., HALPERIN, W.E., and LEMEN, B.A. 1985. Mortality among a cohort of U.S. cadmium production workers - An update. JNCI 74:325-333.

THYSSEN, J., ALTHOFF, J., KIMMERLE, G., and MOHR, U. 1981. Inhalation studies with benzo(a)pyrene in Syrian golden hamsters. J. Natl. Cancer Int. 66:575-577.

TOMATIS, L., and TURUSOV, V. 1975. Studies in the carcinogenicity of DDT. Gann. 17: 219-241.

TOMATIS, L., TURUSOV, V., CHARLES, R.T., BIOCCHI, M., and GATI, E. 1974. Liver tumors in CF-1 mice exposed for limited periods to technical DDT. Z. Krebsforsch. 82:25-35.

TORKELSON, T.R., OYEN, F., MCCOLLISTER, D.D., and ROWE, V.K. 1958. Toxicity of 1,1,1-Trichloroethane as determined in laboratory animals and human subjects. Am. Ind. Hyg. Assoc. J. 19:353-362.

TREON, J., and CLEVELAND, F.P. 1955. Toxicity of certain chlorinated hydrocarbon insecticides for laboratory animals with special reference to aldrin and dieldrin. Agri. Food. Chem. Journ. 3:402.

TSENG, W.P., CHU, H.M., HOW, S.W., FONG, J.M., LIN, C.S., and YEH, S. 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. J. Natl. Cancer Inst. 40:453-463.

TURNER, D.G. 1970. Workbook of Atmospheric Dispersion Estimates. AP-26. U.S. EPA, Office of Air Programs, Research Triangle Park, North Carolina.

TURUSOV, V.S., DAY, N.E., TOMATIS, L., GATI, E., and CHARES, R.T. 1973. Tumors in CF-1 mice exposed for six consecutive generations to DDT. J. Natl. Cancer Inst. 51: 983-998.

TUTE, M.S. 1971. Principles and practice of Hansch analysis: a guide to structure-activity correlation for the medical chemist. Adv. Drug Res. 5:1-77.

TWEEDY, L.D. 1950. Memorandum to Fred Gebhart, Los Angeles County, regarding Special Permit 634. May 10.

VEITH, G.D., DEFOE, D.L., and BERGSTREOT, B.B. 1979. J.Fish Res. Board, Canada 36:1040.

VELSICOL CHEMICAL CORPORATION (VELSICOL). 1955. 2-Year Feeding Study with Rats for Heptachlor. MRID No. 00062599 (Unpublished) (As cited in EPA, 1989a).

VELSICOL CHEMICAL CORPORATION (VELSICOL). 1973. Available from EPA. Toxicity Data for Chlordane. Write to FOI, EPA Washington, D.C., 20460 (as cited in EPA, 1989a).

VERSCHUEREN, K. 1983. Handbook of Environmental Data on Organic Chemicals. Van Nostrand Reinhold Co., New York.

VINOGRADOVA, V.K., KALYAGANOV, P.I., SUDONINA, L.T., and ELIZAROV, G.P. 1973. Hygienic characteristics of working conditions and health status of workers engaged in the production of sodium pentachlorophenolate. (Russ.) Gig. Tr. Prof. Zabol., 8: 11-13

VOGEL, T.M. and McCARTY, P.L. 1985. Biotransformation of tetrachloroethylene to trichloroethylene, dichloroethylene, vinyl chloride, and carbon dioxide under methanogenic conditions. Appl. Environ. Microbiol. 49(5):1080-1083.

VOGEL, T.M., GRIDDLE, C.S., and McCARTY, P.L. 1987. Transformation of Halogenated Aliphatic Compounds. Environmental Science and Technology. Vol. 21 (8):722-735.

VON OETTINGEN, W.F., NEAL, P.A., DONAHUE, D.D., et al. 1942a. The Toxicity and Potential Dangers of Toluene, with Special Reference to its Maximal Permissible Concentration. U.S. Public Health Service Pub., bull. No. 279. p. 50. (As cited in EPA, 1987c).

VON OETTINGEN, W.F., NEAL, P.A., DONAHUE, D.D., et al. 1942b. The toxicity and potential dangers of toluene -- Preliminary Report. J. Am. Med. Assoc. 118:579-584 (As cited in EPA 1987h).

WAHLBERG, J.E. 1968a. Percutaneous Absorption of Radioactive Strontium Chloride Sr89($^{89}SrCl_2$): A Comparison With 11 Other Metal Compounds. Arch. Dermatol. 97:336-339.

WALKER, A.I.T., STEVENSON, D.E., ROBINSON, J., THORPE, E., and ROBERTS, M. 1969. The toxicology and pharmacodynamics of dieldrin (HEOD): Two-Year oral exposure of rats and dogs. Toxicol Appl. Pharmacol. 15:345-373.

WALKER, A.I.T., THORPE, E. and STEVENSON, D.E. 1972. The toxicology of dieldrin (HEOD). I. Long-term oral toxicity studies in mice. Food Cosmet. Toxicol. 11:415-432.

WANG, H.H and MCMAHON, G. 1979. Mortality of workers employed in the manufacture of chlordane and heptachlor. J. Occup. Med. 21:745-748.

WARD, T.E. 1985. Environ. Toxicol. Chem., 4:727-737. (As cited in HSDB, 1989).

WASTE DISPOSAL INC. (WDI). 1955. Letter from F. Caneer to Los Angeles County, Industrial Waste Division, 108 W. 2nd Street, Los Angeles, California. May 27.

WEAST, R.E., ed. 1983. Handbook of Chemistry and Physics. 63rd ed. CRC Press, Cleveland, OH.

WEBSTER, R.C., MOBAYEN, M., and MAIBACH, H.I. 1987. In vivo and in vitro absorption and binding to powered stratum corneum as method to evaluate skin absorpiton of environmental chemical contaminants from ground and surface water. J. Toxicol. Environ. Health 21:367-374.

WIEDEMANN, H.G. 1972. Thermochemochim. Acta. 3:355-366.

WINDHOLTZ, M., BUDAVARI, S., BLUMETTI, R.F., and OTTERBEIN, E.S. 1976. The Merck Index. 9th Edition. Merck and Co., Inc. Rahway, N.J.

WOLF, M.A., ROWE, V.K., McCOLLISTER, D.D., HOLLINGSWORTH, R.C., and OYEN, F. 1956. Toxicological studies of certain alkylated benzenes and benzene. Arch. Ind. Health 14:387-398.

WONG, L.C.K., WINSTON, J.M., HONG, C.B., and PLOTNICK, H. 1982. Carcinogenicity and toxicity of 1,2-dibromoethane in the rat. Toxicol. Appl. Pharmacol. 63:155-165.

WONG, P.T.S., CHAN, Y.K., and LUSON, P.L. 1975. Methylation of lead in the environment. Nature. 253:263-264.

WONG, O., MORGAN, R.W., and WHORTON, M.D. 1983. Comments on the NIOSH Study of Leukemia in Benzene Workers. Technical report submitted to Gulf Canada, Ltd. by Environmental Health Associates.

WORLD HEALTH ORGANIZATION (WHO). 1976. Environmental Health Criteria, Mercury. Geneva.

WORLD HEALTH ORGANIZATION (WHO). 1987. Environmental Health Criteria 71. Pentachlorophenol. Geneva.

YANG, J.J., ROY, T.A. and MICKERER, C.R. 1986a. Percutaneous absorption of benzo(a)pyrene in the rat. Toxicol. and Indust. Health 2:409-415.

YANG, J.J., ROY, T.A. and MICKERER, C.R. 1986b. Percutaneous absorption of anthracene in the rat. Toxicol. and Indust. Health 2:79-84.

YANG, G., WANG, S., ZHOU, R., and SUN, S. 1983. Endemic selenium intoxication of humans in China. Am. J. clin. Nutr. 37:872-881.

YORK, R.G., SOWRY, B., HASTINGS, L., and MANSON, J. 1982. Evaluation of teratogenicity and neurotoxicity with maternal inhalation exposure to methyl chloroform. J. Toxicol. Environ. Health. 9:251-266. (As cited in EPA, 1984i).

ZOECON CORPORATION. 1983. MRID No. 00128356. As cited in EPA, 1989a. Available from EPA.

APPENDIX A

TOXICITY PROFILES FOR SELECTED CHEMICALS

WASTE DISPOSAL, INC.

TABLE OF CONTENTS

APPENDIX A

A.1 ARSENIC

INTRODUCTION

Arsenic is a naturally occurring metalloid which can be present in a number of different valence states and as a constituent of both inorganic and organic compounds. Elemental arsenic is used in industry as an alloying agent; both inorganic and organic arsenical compounds have been used as pesticides (ACGIH, 1986). The average concentration of arsenic in U.S. drinking water is approximately 2 μg/liter (Greathouse and Craun, 1978). U.S. surface water and groundwater surveys indicate that over 90% of all surface water contains 10 μg/liter or less and average concentrations in well water are generally less than 20 μg/liter (ATSDR, 1987). U.S. ambient air concentrations have been estimated to be in the range of 2.6-10.9 ng/m^3 (Akland, 1983).

TOXICOKINETICS

Absorption of orally administered arsenic is a function of the solubility of the specific compound administered and the dosing rate. Soluble inorganic arsenic is rapidly and almost completely absorbed from the gastrointestinal tract in rats (Coulson et al., 1935). Estimates by Coulson et al. (1935) and Ray-Bettley and O'Shea (1975) indicate that greater than 95% of ingested inorganic arsenic is absorbed by man. Absorption of inhaled arsenic, in the form of an aerosol or a dust, is dependent upon particle size. Particles smaller than 7-8 um in diameter may deposit deep in the lungs and be absorbed by the respiratory epithelium. Larger particles are deposited primarily in the upper airways, cleared from the lung by mucociliary action and swallowed (EPA, 1984b). Once absorbed, arsenic is widely distributed. The highest concentrations occur in nails, hair, bone, teeth and skin with lower levels in the heart, kidney, liver and lung. Long-term accumulation of arsenic generally does not occur in physiologically vital body organs. Excretion is predominantly through the urine (EPA, 1984a).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

CARCINOGENIC POTENTIAL

Epidemiological studies have demonstrated that inhalation of arsenic is strongly associated with lung cancer in occupationally exposed workers (Enterline et al., 1987; Lee-Feldstein, 1983) and in nonoccupationally exposed individuals living near arsenic-emitting smelters (Brown et al., 1984). Ingestion of arsenic has been linked to skin cancer (Tseng et al., 1968) and more recently to bladder, liver, and lung cancer (Chen et al., 1986). Although arsenic's potential as a human carcinogen has long been recognized, it is only recently that its carcinogenicity has been demonstrated in animal models. Intratracheal administration of arsenic trioxide (Ishinishi et al., 1983) or calcium arsenate (Pershagen, 1985; Pershagen and Bjorklund, 1985) has been reported to increase lung tumors in hamsters. Attempts to induce tumors in laboratory animals following oral exposure have generally been inconclusive or negative (EPA, 1986a).

GENOTOXIC POTENTIAL

Arsenic has been tested for gene mutations in bacteria, yeast and cultured mammalian cells. Negative results have been reported in almost all tests (ATSDR, 1987). Weakly positive results were reported for sodium arsenite in a reverse mutation assay in Saccharomyces cerevisiae (Singh, 1983). After review of these assays, EPA concluded that arsenic is either inactive or an extremely weak inducer of gene mutations (Jacobson-Kram and Montalbano, 1985).

In contrast, arsenic has been reported to induce chromosome aberrations and sister-chromatid exchanges in cultured mammalian cells (Petres et al., 1970, 1972; Jacobson-Kram and Montalbano, 1985; Petres and Hundeiker, 1968) and in circulating lymphocytes from individuals exposed in vivo (Burgdof et al., 1977; Nordenson et al., 1979; Petres et al., 1970). EPA (1984a) noted

that results from tests in human circulating lymphocytes must be interpreted with caution because these studies are limited by methodological difficulties including small sample sizes and likely exposure of subjects to other clastogenic agents.

REPRODUCTIVE EFFECTS

Arsenic has been observed to produce fetotoxic and developmental effects in the offspring of exposed rats, mice and hamsters. Intraperitoneal injection of 10-45 mg/kg/day sodium arsenate has been reported to increase the numbers of fetal malformations in several studies (ATSDR, 1987). Oral administration of 120 mg/kg sodium arsenate to pregnant mice by gavage was observed to produce decreased fetal birth weight and increased prenatal mortality; no effects were observed with doses below 100 mg/kg (Hood et al., 1977, 1978). Oral administration of sodium arsenite has also been reported to produce the same fetotoxic effects in the offspring of pregnant mice exposed to 40 or 45 mg/kg (Hood and Harrison, 1982). Signs of maternal toxicity were reported at these dose levels in both studies.

ACUTE/CHRONIC EFFECTS

Information on the toxic effects of arsenic has been reviewed by EPA (1980, 1984a) and more recently by the Agency for Toxic Substances and Disease Registry (ATSDR, 1987). Acute poisoning in humans may result in gastrointestinal disturbances (nausea, vomiting and diarrhea), hemolysis and encephalopathy. Chronic exposure produces peripheral neuropathy characterized by paresthesia, hyperesthesia, neuralgia and muscular pain and weakness. Other indicators of central neuropathy include mental retardation in children, hearing loss and abnormal electroencephalograms. Toxic effects reported in other systems following chronic arsenic exposure include dermal keratoses and hyperpigmentation, precancerous dermal lesions, and cardiovascular, hepatic and renal injury.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

EPA (1984a) classified arsenic in Group A--Human Carcinogen. This category applies to chemical agents for which there is sufficient evidence of carcinogenicity from animal and human studies.

INGESTION

To estimate the risks posed by ingestion of arsenic, EPA (1984a) used the epidemiological data reported by Tseng et al. (1968) for a Taiwanese population exposed to arsenic at a concentration of approximately 0.4-0.6 mg/liter in drinking water. In 37 villages that had obtained drinking water from artesian wells with elevated levels of arsenic for 45 years, 40,421 individuals were examined for hyperpigmentation, keratosis, skin cancer, and blackfoot disease (in which impaired peripheral circulation leads to gangrene). The local well waters were analyzed for arsenic, and age-specific cancer prevalence rates were found to be correlated with both local arsenic concentrations and age (or duration of exposure). EPA (1984a) extrapolated to low dose levels and estimated an oral carcinogenic potency factor of 15 $(mg/kg/day)^{-1}$. In the same area, Chen et al. (1986) reported an association between bladder, lung, and liver tumors and ingestion of arsenic-contaminated drinking water.

The Risk Assessment Forum of EPA (1988) recently reassessed the carcinogenicity risks associated with ingestion of inorganic arsenic, and concluded that the Tseng (1977) study, a reevaluation of the 1968 study, was the most appropriate for estimating quantitative risk values. The study reported an increased prevalence of skin cancer in humans exposed to arsenic in the drinking water. A revised cancer potency factor of 2 $(mg/kg/day)^{-1}$ was proposed. The possibility that various internal tumors may also be associated with oral arsenic exposure was not taken into consideration in this revised estimate, and further review of the epidemiological studies supporting this possibility is underway at EPA.

One factor complicating risk assessments for ingested arsenic is possible variation in carcinogenic potency according to the valence state of arsenic. Trivalent inorganic arsenic compounds are generally more toxic than pentavalent inorganic arsenic compounds or organic arsenic compounds (EPA, 1980, 1984a), and epidemiological studies of copper smelter workers have often reported that increased lung cancer incidences were associated with exposure to trivalent inorganic arsenic. For these reasons, EPA (1980) speculated that the carcinogenic potency factor for ingested arsenic may be applicable only to trivalent inorganic arsenic. However, recent studies have demonstrated water samples from the area of Taiwan where the epidemiological study reported by Tseng et al. (1968) was conducted contain primarily pentavalent inorganic arsenic and no organic arsenicals (EPA, 1984a).

In addition to the oral carcinogenic potency factor, EPA (1989) reported an oral RfD of $1x10^{-3}$ mg/kg/day. The RfD is also based on the Tseng (1977) study described above. An uncertainty factor of 1 was used to derive the RfD.

INHALATION

The health risks posed by airborne arsenic compounds have been reviewed in considerable detail by EPA (1984a), and studies on the carcinogenicity of arsenic compounds were reviewed by the International Agency for Research on Cancer (IARC) in 1980. Risk assessments for exposure to airborne arsenic were presented by the Occupational Safety and Health Administration (OSHA, 1983) and EPA (1984a). The following summary is based on these reviews and risk assessments and on an extensive review of the primary literature.

EPA (1984a) based its quantitative risk assessment for inhaled arsenic on five studies of three exposed worker populations (Higgins et al., 1982; Lee-Feldstein, 1983; Brown and Chu, 1982, 1983a,b; Enterline and Marsh, 1980; Ott et al., 1974). All five studies reported excess lung cancer risks that were related to the intensity and duration of exposure and the duration of

follow-up (latency). The estimates of unit risk[1] obtained from the five studies were in reasonably good agreement, ranging from 1.25×10^{-3} to 1.36×10^{-2} $(\mu g/m^3)^{-1}$. EPA omitted the highest value, which was considered the least reliable, and calculated the geometric mean for each of the two remaining populations and then an overall geometric mean to obtain a best estimate of 4.3×10^{-3} $(\mu g/m^3)^{-1}$ for the unit risk. EPA (1984a) applied the assumptions that humans weigh 70 kg, inhale 20 m^3 of air per day and absorb 30% of inhaled arsenic to this unit risk to calculate a carcinogenic potency factor for inhaled arsenic of 50 mg/kg/day.

The unit risk value derived from these studies is thus an estimate of the unit risk for exposure to the high levels characteristic of the three work environments where the data were obtained. This value is intended to be an upper-limit estimate of risk, and actual risks are unlikely to be higher but may be lower than this estimated value.

The EPA primary drinking water standard for arsenic is 50 μg/liter (CFR, 1984). This value was established as a maximum allowable level for arsenic in drinking water by the U.S. Public Health Service in 1942, and it continues to be used in the current EPA regulations (EPA, 1985). EPA's Office of Drinking Water is considering maintaining the present maximum contaminant level for arsenic in municipal drinking water supplies (EPA, 1985), and has proposed a maximum contaminant level goal (MCLG) of 50 μg/liter.

The American Conference of Governmental Industrial Hygienists (ACGIH, 1986) recommends a time-weighted average threshold limit value (TLV) of 0.2 mg/m^3 for arsenic and soluble compounds of arsenic.

---

[1] The unit risk is the risk associated with lifetime exposure to 1 unit (generally 1 mg/kg/day, 1 mg/liter, or 1 ug/m^3) of a substance.

SUMMARY OF ARSENIC CRITERIA

EPA carcinogen classification	Group A
Oral carcinogenic potency factor	2 $(mg/kg/day)^{-1}$
Inhalation carcinogenic potency factor	50 $(mg/kg/day)^{-1}$
Oral RfD	$1x10^{-3}$ mg/kg/day
MCL	50 μg/liter
MCLG	50 μg/liter

REFERENCES

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987. Draft Toxcological Profile for Arsenic. U.S. Public Health Service.

AKLAND, G. 1983. Memo to D. Sivulka, dated February 23, 1983. Available from: U.S. Environmental Protection Agency, Research Triangle Park, NC; Project file No. ECAO-HA-79-5.

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio.

ANDELMAN, J.B., and BARNETT, M. 1985. Feasibility Study to Resolve Questions on the Relationship of Arsenic in Drinking Water to Skin Cancer. U.S. Environmental Protection Agency. Cooperative Agreement No. CR-806815-02-1.

ASTOLFI, E., MACCAGNO, A., GARCIA-FERNANDEZ, J.C., VACCARO, R., and STIMOLA, R. 1981. Relation between arsenic in drinking water and skin cancer. Biol. Trace Elem. Res. 3:133-143.

BAXLEY, M.N., HOOD, R.D., VEDEL, G.C., HARRISON, W.P., and SZCZECH, G.M. 1981. Prenatal toxicity of orally administered sodium arsenite in mice. Bull Environ Contam Toxicol 26:749-756 (As cited in EPA 1984b).

BROWN, C.C., and CHU, K.C. 1982. Approaches to epidemiologic analysis of prospective and retrospective studies: Example of lung cancer and exposure to arsenic. In Prentice, R.L., and Whittemore, A.S., (eds.) Environmental Epidemiology: Risk Assessment. SIAM, Philadelphia.

BROWN, C.C., and CHU, K.C. 1983a. A new method for the analyses of cohort studies: Implications of the multistage theory of carcinogenesis applied to occupational arsenic exposure. Environ. Health Perspect. 50:293-308.

BROWN, C.C., and CHU, K.C. 1983b. Implications of the multistage theory of carcinogenesis applied to occupational arsenic exposure. JNCI 70:455-463.

BROWN, L.M. POTTERN, L.M. and BLOT, W.J. 1984. Lung cancer in relation to environmental pollutants emitted from industrial sources. Environ. Res. 34:250-261.

BURGDORF, W., KURVINK, K. and CERVENKA, J. 1977. Elevated sister chromatid exchange rate in lymphocytes of subjects treated with arsenic. Hum Genet. 36:69-72.

CARLSON, E.J. et al., 1935. Metabolism in the rat of the naturally occurring arsenic of shrimp as compared with arsenic trioxide. J. Nutr. 10:255-270 (As cited in EPA 1984a).

CHEN, C., CHUANG, Y., YOU, S., LIN, T., and WU, H. 1986. A retrospective study on malignant neoplasms of bladder, lung and liver in blackfoot disease endemic area in Taiwan. Brit. J. Cancer 53:399-405.

CODE OF FEDERAL REGULATIONS (CFR). 1984. 40 CFR 141, Environmental Protection Agency, National Interim Primary Drinking Water Regulations. U.S. Government Printing Office, Washington, D.C.

COULSON, E.J., REMINGTON, R.E., and LYNCH, K.M. 1935. Metabolism in the rat of the naturally occuring arsenic of shrimp as compared with arsenic trioxide. J. Nutr. 10: 255-270.

COX, D.P. 1975, Methylation of Arsenic, IN: Arsenical Pesticides, ACS Symposium Series, R.F. Gould, ed.

ENTERLINE, P.E., and MARSH, G.M. 1980. Mortality studies on smelter workers. J. Ind. Med. 1:251-259.

ENTERLINE, P.E., HENDERSON, V.L. and MARCH, G.M. 1987. Exposure to arsenic and respiratory cancer. A reanalysis. Am. J. Epidemiol. 125(6):929-938.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1979. Water-Related Envioronmental Fate of 129 Priority Pollutants. December. Washington, D.C. EPA 440/4-79-029.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Arsenic. Office of Water Regulations and Standards, Criteria and Standards Division. October. Washington, D.C. EPA 440/5-80-021.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984a. Health Assessment Document for Inorganic Arsenic. Office of Health and Environmental Assessment, March. Washington, D.C. EPA 600/8-83-021F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984b. Health Effects Assessment for Asenic. Environmental Criteria and Assessment Office. September. Cincinati, OH.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. National primary drinking water regulations; synthetic organic chemicals, inorganic chemicals and microorganisms; proposed rule. November 13. 40 CFR Part 141. Fed. Reg. 50(219):46936-47025.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986a. Special Report on Ingested Inorganic Arsenic and Certain Human Health Effects. External Review Draft. Risk Assessment Forum. October. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986b. Superfund Public Health Evaluation Manual. Office of Emergency and Remedial Response, October. Washington, D.C. EPA 540/1-86-060.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Updated reference dose and Cancer Potency Numbers for use in Risk Assessment. November 16. Memo from Sandra Lee Program Manager, Toxics Integration Branch.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Special Report on Ingested Inorganic Arsenic Skin Cancer; Nutritional Essentiality. Risk Assessment Forum, U.S. EPA, Washington D.C.

GREATHOUSE, D.G. and CRAUN, G.F. 1978. Cardiovascular disease study - Occurrence of inorganics in household tap water and relationships to cardiovascular mortality rates. In: Hemphill, D.D. (ed). Proceedings of the Trace Substance in Environmental Health Conference, Columbia, MO. University of Missouri. 12:31-39.

HIGGINS, I., WELCH, K., and BURCHFIELD, C. 1982. Mortality of Anaconda smelter workers in relation to arsenic and other exposures. Dept. of Epidem., Univ.of Michigan, Ann Arbor, Michigan (As cited in EPA 1984a).

HOOD, R.D., HARRISON, W.P. 1982. Effects of prenatal arsenite exposure in hamster. Bull. Environ. Contam. Toxicol. 29:671-678 (As cited in ATSDR, 1987).

HOOD, R.D., THACKER, G.T. PATTERSON, B.L., SZCZECH, G.M. 1978. Prenatal effects of oral versus intraperitoneal sodium arsenate in mice. J. Environ. Pathol. Toxicol. 1:857-864.

HOOD, R.D., THACKER, G.T., PATTERSON, B.L. 1977. Effects in the mouse and rat of prenatal exposure to arsenic. Environ. Health Perspect. 19:219-222.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1980. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Vol. 23: Some Metals and Metallic Compounds. World Health Organization, Lyon, France. Pp. 39-324.

ISHINISHI, N., YAMAMOTO, A., HISANAGA, A., and INAMASU, T. 1983. Tumorigenicity of arsenic trioxide to the lung in Syrian golden hamsters by intermittent instillations. Cancer Lett. 21:141-147.

JACOBSON-KRAM, D., MONTALBANO, D. 1985. The reproductive effects assessment groups' report on the mutagenicity of inorganic arsenic. Environ. Mutagen. 7:787-804.

LEE-FELDSTEIN, A. 1983. Arsenic and respiratory cancer in man: Follow-up of an occupational study. In Lederer, W., and Fensterheim, R., (eds.) Arsenic: Industrial, Biomedical and Environmental Perspectives. Van Nostrand Reinhold, New York.

MATSUMOTO, N. OKINO, T., KATSUNUMA, H. and IIJIMA, S. 1973b. Effects of Na-arsenite on the growth and development of the foetol mice. Congenit Anom Curr Lit; 13:175-176 (As cited in EPA 1984b).

MATSUMOTO, N. OKINO, T., KATSUNUMA, H. and IIJIMA, S. 1973a. Effects of Na-arsenate on the growth and development of foetal mice. Teratology; 8:98 (As cited in EPA, 1984b).

NORDENSON, I., SALMONSSON, S., BRUN, E., BECHMAN, G., 1979. Chromosome aberrations in psoriatic patients treated with arsenic. Hum. Genet. 48:1-8 (As cited in ATSDR, 1987).

OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION (OSHA). 1983. Occupational exposure to inorganic arsenic. Fed. Reg. 48:1864-1903.

OTT, M.G., HOLDER, B.B., and GORDON, H.L. 1974. Respiratory cancer and occupational exposure to arsenicals. Arch. Environ. Health 29:250-255.

PERSHAGEN, G. 1985. Lung cancer mortality among men living near an arsenic-emitting smelter. Am. J. Epidemiol. 122:684-696.

PERSHAGEN, G. and BJORKLUND, N.E. 1985. On the pulmonary tumorgenicity of arsenic trisulfide and calcium arsenate in hamsters. Cancer Letters. 27:99-104.

PERSHAGEN, G., NORDBERG, G., and BJORKLUND, N.E. 1983. Carcinomas of the respiratory tract in hamsters given arsenic trioxide and/or benzo(a)pyrene by the pulmonary route. Environ. Res. (In Press). (As cited in EPA, 1984b).

PETRES, J. and HUNDEIKER, M. 1968. "Chromosomenpulverisation" nach Arseneinwir Kung auf Zellkulburen in vitro. Arch Klin Exp Dermatol; 231:366 (As cited in EPA, 1984b).

PETRES, J., et al. 1970. Chromosomenaberrationen an menschlichen Lymphozyken bei chronischen Arsenschaden. Dtsh Med Wochenschr; 95:79 (As cited in EPA 1984b).

PETRES, J., et al. 1972. Zum Einfluss anorganischen Arsens auf die DNS-Synthese menschlicher Lumphocyten in vitro. Arch Derm Forch; 242:343 (As cited in EPA 1984b).

RAY-BETTLEY, F. and O'SHEA, J.A. 1975. The absorption of arsenic and its relation to carcinoma. Br. J. Dermatol. 96:563 (As cited in EPA 1980).

SINGH, I. 1983. Induction of reverse mutation and mitotic gene conversion by some metal compounds in Saccharomyces cerevisiae. Mutat Res. 117:149-152.

TSENG, W.P., CHU, H.M., HOW, S.W., FONG, J.M., LIN, C.S., and YEH, S. 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. J. Natl. Cancer Inst. 40:453-463.

TSENG, W.D. 1977. Effect and Dose-response relationships of skin cancer and blackfoot disease with arsenic. Environ. Health Perspect. 19:009-119.

A.2 BENZENE

INTRODUCTION

Benzene is a volatile, colorless, flammable liquid aromatic hydrocarbon with a characteristic odor. It is a chemical intermediate in the synthesis of compounds such as styrene, synthetic rubber, and phenol, and it is used as a gasoline additive to increase the octane rating.

Releases of benzene to the environment are usually to air, due to its volatility, with smaller amounts released to water and soil. Benzene concentrations in urban air have been estimated to range from 0.02-65 ppb with the majority of mean concentrations below 5 ppb (ATSDR, 1987). Benzene levels in indoor air are often greater than levels found in outdoor air. The highest concentration reported in federal drinking water surveys is 80 μg/liter. Approximately 1.3% of all groundwater systems are estimated to contain benzene at concentrations greater than 0.5 μg/liter; approximately 3% of all surface water systems are estimated to contain benzene at levels greater than 0.5 μg/liter with none expected to exceed 5 μg/liter (EPA, 1987b).

TOXICOKINETICS

Benzene is readily absorbed into the body via ingestion and inhalation. Dermal absorption is somewhat slower. It is stored in the bone marrow, liver, and body fat. Elimination of benzene occurs via exhalation of unchanged benzene through the lungs or by metabolism in the liver with subsequent excretion of metabolites in the urine (EPA, 1985a). Major urinary metabolites are phenolic compounds (e.g., phenol, catechol, hydroquinone). Conjugated phenolic metabolites appear in the urine mainly as etheral sulphates and glucuronides (IARC, 1982b).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

CARCINOGENIC POTENTIAL

Several epidemiological studies have described a causal relationship between exposure to benzene (concentrations unspecified) by inhalation (either alone or in combination with other chemicals) and leukemia in humans (IARC, 1982a,b). Dose levels associated with the carcinogenic response have not been firmly established. Some studies suggest that low levels may be carcinogenic, but this is not universally accepted (ATSDR, 1987). Most cases observed were acute myelogenous leukemias, although some were monocytic, erythroblastic, or lymphocytic. Various hematological disorders other than leukemia have also been reported including aplastic anemia and pancytopenia (IARC, 1982b). A series of epidemiological studies, both cohort and case-control, showed statistically significant associations between leukemia and occupational exposure (concentration unspecified) to benzene (Askoy et al., 1985; Wong, 1982; Rinsky et al., 1987; Ott et al., 1978). These results have been replicated in a number of countries and in different industries (IARC, 1982b).

The carcinogenicity of benzene has been evaluated in rats and mice by various routes of exposure (inhalation, oral, dermal, subcutaneous). Most earlier studies were negative. Maltoni et al. (1979) first reported increased incidences of mammary gland, Zymbal gland, and skin carcinomas, leukemias, angiosarcomas and liver tumors in rats exposed by gavage. Recently, oral exposure has been associated with increased incidences of Zymbal gland and mammary gland carcinomas in a second study (NTP, 1986). Inhalation exposure to benzene has been associated with thymic lymphoma, hematopoietic neoplasms, and leukemia (Cronkite et al., 1985; Snyder et al., 1980). Studies involving dermal exposure have been negative for carcinogenicity (IARC, 1982b). Leukemia has been observed in studies in which benzene was administered by subcutaneous injection; however, these studies were limited in some instances by lack of control groups and in others by high incidences of leukemia in untreated controls (IARC, 1982b).

GENOTOXIC POTENTIAL

The genotoxicity of benzene has been reviewed by IARC (1982b) and ATSDR (1987). Benzene does not induce gene mutations in bacterial systems. It has not been found to be a point mutagen in mammalian cells; however, benzene did induce cytogenetic abnormalities in mammalian cells in vitro (chromosomal aberrations and sister-chromatid exchanges). Benzene causes both structural and numerical chromosome aberrations in vivo in humans and animals, and in vitro in cultured cells (ATSDR, 1987). Several studies demonstrated that benzene exposure of experimental animals in vivo induces chromosomal aberrations in bone marrow cells. There is a clear correlation between exposure to benzene and the appearance of chromosomal aberrations in the bone marrow and in peripheral lymphocytes of individuals exposed to high levels of benzene (more than 100 ppm) (IARC, 1982b).

REPRODUCTIVE EFFECTS

Inhalation experiments conducted in rats, mice, guinea pigs, and rabbits suggest that benzene is not teratogenic at doses that are fetotoxic and embryolethal (IARC, 1982b). It has been shown to be embryo/fetotoxic at maternally toxic dose levels and it is a potent inhibitor of growth and development in utero (EPA, 1985a). Increased incidences of fetal resorptions, skeletal variations and altered fetal hematopoiesis have been reported (ATSDR, 1987).

Animal experiments in rats, guinea pigs, and rabbits suggest that exposures to benzene vapors may damage the testes of adult males (IARC, 1982b).

ACUTE/CHRONIC EFFECTS

Oral LD_{50} values for reagent-grade benzene in male Sprague-Dawley rats are reported to range from 0.93 g/kg to 4.9 g/kg. An oral LD_{50} of 5.6 g/kg was reported in male Wistar rats (IARC, 1982b). An oral LD_{50} of 4.7 g/kg has

been reported for the mouse (Sandmeyer, 1981). These LD_{50} values suggest that benzene is a slightly toxic compound following acute exposure (Hodge and Steiner, 1949).

The toxic effects of benzene vapors in humans and other animals include hematological, immunological and central nervous system effects (EPA, 1985a).

Hematological System

Acute exposure to benzene is generally not associated with hematotoxicity in humans or animals (ATSDR, 1987). Chronic human exposure can cause a continuum of changes in the circulatory formed blood elements and bone marrow precursors (EPA, 1985a). Leucopenia, thrombocytopenia, anemia, or combinations of these may occur. At early stages of such blood dyscrasias, these effects appear to be reversible. Exposure for longer periods of time may lead to pancytopenia, which results from bone marrow toxicity and is considered to be irreversible (IARC, 1982b). Leucopenia is the most commonly observed effect of chronic benzene intoxication in laboratory animals. Longer exposure periods may lead to pancytopenia and bone marrow depression (EPA, 1985a).

Immune System

Immune system depression by benzene is well known. Depression of serum antibodies (IgG and IgA) in benzene workers (exposure concentration unspecified) has been reported (EPA, 1985a). In addition, administration of benzene to mice *in vivo* inhibits the function of B- and T-lymphocytes *in vitro* (IARC, 1982b). These observations, as well as the recognized ability of benzene to depress leukocytes, may explain why benzene-exposed individuals readily succumb to infection and the terminal event in severe benzene toxicity is often overwhelming infection (IARC, 1982b).

Central Nervous System

In humans, acute exposures to high levels of benzene vapors (20,000 ppm) produce central nervous system effects including dizziness, giddiness, exhilaration, nausea, vomiting, headache, drowsiness, staggering, loss of balance, narcosis, coma and death. Exposure to 25,000 ppm is rapidly fatal; death is usually the result of respiratory or cardiac failure (NAS, 1976). Neuritis and other neurological effects may also be produced by chronic exposure to lower doses (Sandmeyer, 1981). In experimental animals, acute exposure to high concentrations of benzene vapors may cause central nervous system depression and death.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

Applying EPA's criteria for evaluating the overall weight of evidence of carcinogenicity to humans (EPA, 1986a), benzene has been classified in Group A-Human Carcinogen. This category indicates that there is sufficient evidence from epidemiological studies to support a causal association between an agent and cancer. The International Agency for Research on Cancer (IARC) has also concluded that there is sufficient evidence that benzene is carcinogenic in humans following inhalation (IARC, 1982a).

The EPA (1986b) Carcinogen Assessment Group (CAG) calculated a carcinogenic potency factor for benzene derived from human epidemiological studies (Ott et al., 1978; Rinsky et al., 1981) in which significantly increased incidences of leukemia were observed in workers exposed to benzene principally by inhalation. On the basis of these data, EPA estimated an inhalation cancer potency factor of 2.6×10^{-2} $(ppm)^{-1}$ or 2.9×10^{-2} $(mg/kg/day)^{-1}$. A cancer potency factor of 2.9×10^{-2} $(mg/kg/day)^{-1}$ for oral exposure was also derived by EPA (1988) on the basis of these epidemiological studies. These cancer potency factors are 95% upper-bound estimates; the true potency factors are unlikely to be greater, but may be lower than these estimates.

EPA (1985b) promulgated a final drinking water maximum contaminant level goal (MCLG) of zero because benzene is a human carcinogen. EPA (1987b) also established a final drinking water maximum contaminant level (MCL) of 5 μg/liter. MCLGs take into consideration health effects only, while MCLs take into consideration analytic procedures, treatability, occurrence, and cost, in addition to health effects.

The EPA Office of Drinking Water developed a ten-day health advisory (HA) of 235 μg/liter for children (EPA, 1987a). The HA was based on an inhalation study in which 44 ppm caused depressed white blood cell counts in Sprague-Dawley rats within 5-8 weeks (Deichmann et al., 1963). Doses of 29 or 31 ppm had no effect after a four month exposure. The 31 ppm (96 mg/m^3) dose was identified as the no-observed-adverse effect level (NOAEL) for this study. Health advisories for longer exposure periods were not developed because of the potent carcinogenic response of benzene (EPA, 1987a).

The American Conference of Governmental Industrial Hygienists (ACGIH, 1986) has recommended a time-weighted average threshold limit value of 10 ppm (approximately 30 mg/m^3) for occupational exposure to benzene. It was also specified that benzene should not be employed when substitute materials are available. The TWA-TLV was lowered to 1 ppm by OSHA in 1987 (FR, 1987), but was recently reinstated at 10 ppm by OSHA in 1989 (OSHA, 1989).

SUMMARY OF BENZENE CRITERIA

EPA carcinogen classification	Group A
Oral carcinogenic potency factor	2.9×10^{-2} $(mg/kg/day)^{-1}$
Inhalation carcinogenic potency factor	2.9×10^{-2} $(mg/kg/day)^{-1}$
MCL	5 μg/liter
MCLG	0 μg/liter
Ten-day HA (child)	235 μg/liter

REFERENCES

AGENCY FOR TOXIC SUBSTANCE and DISEASE REGISTRY (ATSDR). 1987. Draft Toxicological Profile for Benzene; U.S. Public Health Service.

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio

ASKOY, M. 1985. Malignancies due to occupational exposure to benzene. Am. J. Ind. Med. 7:395-402

CRONKITE, E.P., DREW, R.T., INONE, T., and BULLIS, J.E. 1985. Benzene hematotoxicity and leukemogenesis. Am. J. Ind. Med. 7:447-456

DEICHMANN, W.G., MACDONALD, W.E., and BERNAL, E. 1963. The hemopoietic tissue toxicity of benzene vapors. Toxicol. Appl. Pharmacol. 5:201-224. (As cited in EPA, 1987a).

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Washington, D.C. December 1979. EPA 440/4-79-029

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Benzene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. EPA 40/5-80-0018

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Benzene. Environmental Criteria and Assessment Office. Cincinnati, Ohio. September 1984. EPA 540/1-86-037

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Drinking Water Criteria Document for Benzene (Final Draft). Office of Drinking Water, Washington, D.C. April 1985

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. National primary drinking water regulations; volatile synthetic organic chemicals, final rule. Fed. Reg. 50:46880-46901 (November 13, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986a. Guidelines for carcinogen risk assessment. Fed. Reg. 51:33992-34003 (September 24, 1986)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986b. Evaluation of the Potential Carcinogenicity of Benzene (71-43-2). Prepared by Carcinogen Assessment Group for Office of Emergency and Remedial Response and Office of Solid Waste and Emergency Response. Washington, D.C. OHEA-C-073-29

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986c. Superfund Public Health Evaluation Manual. Office of Emergency and Remedial Response. Washington, D.C. October 1986

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987a. Draft Health Advisory for Benzene. Office of Drinking Water, Washington, D.C. March 31, 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987b. National primary drinking water regulations; synthetic organic chemicals; monitoring for unregulated contaminants; final rule. Fed. Reg. 52:25690-25717 (July 8, 1987)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Integrated Risk Information System (IRIS). On-line 03/01/88.

FEDERAL REGISTER (FR). 1987. Department of Labor; Occupational Safety and Health Administration. 29 CFR Part 1910; Occupational Exposure to Benzene; Final Rule. Vol. 52, No. 176.

GAFFNEY, J.S. and LEVINE, S.Z. 1979. Predicting gas phase organic molecular reaction rates using linear free-energy correlation. I. $O(^3P)$ and OH addition and abstraction reactions. Int. J. Chem. Kinet. XI: 1197-1209

HODGE, H.C. and STEINER, J.H. 1949. Tabulation of Toxicity Classes. Am. Ind. Hyg. Assoc. Q. 10:93-96

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1982a. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Chemicals, industrial processes and industries associated with cancer in humans. Supplement 4 pp. 56-57

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1982b. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans Some industrial Chemicals and dyestuffs. Volume 29:93-148

MACKAY, D. and LEINONEN, P.J. 1975. Rate of evaporation of low-solubility contaminants from water bodies to atmosphere. Environ. Sci. Technol. 9(13): 1178-1180

MALTONI, C. and SCARNATO, C. 1979. First experimental demonstration of the carcinogenic effects of benzene. Med. Lauoro 70:352-357.

NATIONAL ACADEMY OF SCIENCE (NAS). 1976. Health Effects of Benzene: A Review Committee on Toxicology, Assembly of Life Sciences. National Research Council, Washington, D.C.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1986. Toxicology and Carcinogenesis Studies of Benzene (CAS No. 71-43-2) in F344/N Rats and B6C3F_1 Mice (Gavage Studies). Technical Report Series No. 289. NIH Publication No. 86-2545

OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION (OSHA). 1989. Air Contaminants – Permissible Exposure Limits. Table Z-2.

OTT, M.G., TOWNSEND, J.C., FISHBECK, W.A., and LANGER, R.A. 1978. Mortality among individuals occupationally exposed to benzene. Arch. Environ. Health 33:3-10

RINSKY, R.A., YOUNG, R.J., and SMITH, A.B. 1981. Leukemia in benzene workers. Am. J. Med. 3:216-245

RINSKY, R.A., SMITH, A.B., HORNUNG, R., FILLOON, T.G., YOUNG, R.J., OKUN, A.H., and LANDRIGAN, P.J. 1987. Benzene and Leukemia: An Epidemiologic Risk Assessment. N. Eng. J. Med. 316:1044-1050

SANDMEYER, E.E. 1981. Aromatic hydrocarbons in: Patty's industrial hygiene and toxicology, vol 2, 3rd rev. ed., Clayton, G.D., Clayton F.E., eds. New York: Interscience Publishers: pp. 3253-3283

SNYDER, C.A., GOLDSTEIN, B.D., SELLAKUMAR, A.R., BROMBERG, I., LASKIN, S., and ALBERT, R.E. 1980. The inhalation toxicology of benzene: Incidence of Hematopoietic neoplasms and hematotoxicity in AKR/J and C57BL/6J mice. Toxicol. Appl. Pharmacol. 54:323-331

VERSCHUEREN, K. 1977. Handbook of Environmental Data on Organic Chemicals. Van Nostrand Reinhold Co., New York.

WEAST, R.E., ed. 1983. Handbook of Chemistry and Physics. 63rd ed. CRC Press, Cleveland, Ohio.

WONG, O. 1982. An Industry-wide Mortality Study of Chemical Workers Occupationally Exposed to Benzene. Prepared for the Chemical Manufacturers Association by Environmental Health Associates, Oakland, California

A.3 CADMIUM

INTRODUCTION

Cadmium is an element that occurs widely in nature, usually in lead or zinc ores. It is produced as a by-product during processing of these ores. Elemental cadmium is insoluble in water, although many cadmium salts are quite soluble (EPA, 1985a,b). Cadmium is used in metal plating, pigments, batteries, plastic stabilizers and other uses. The general human population is exposed to cadmium in drinking water and food; cigarette smoke also contains high levels of cadmium. Additional inhalation exposure occurs in industrial settings (EPA, 1985a,b).

Cadmium is found naturally in surface water and groundwater at concentrations in the range of 1 - 10 μg/l (EPA, 1987a). A recent survey of Canadian drinking waters reported an arithmetic mean cadmium concentration of 0.05 μg/liter (Meranger et al., 1981). Ambient air concentrations estimated by Friberg et al. (1974) were generally below 3 ng/m^3 in rural areas and ranged up to 50 ng/m^3 in industrialized areas.

TOXICOKINETICS

Cadmium is poorly absorbed (about 1-6% of an administered dose) following oral exposure (ATSDR, 1987). Gastrointestinal absorption is influenced by many factors including age, diet, dose and concomitant exposure to other metals (ATSDR, 1987); in general, young animals absorb more than adults (Sasser and Jarboe, 1977; Kostial et al., 1978) and females absorb more than males (Sumino et al., 1975). Cadmium is more readily absorbed following inhalation (about 30-60% of the inhaled dose) and absorption depends on the size of the inhaled particle (ATSDR, 1987). Only small quantities are absorbed through intact skin (Wahlberg, 1965). Cadmium distributes throughout the body with the highest concentrations located in the liver and kidney in rats (Sabbioni et al., 1978) and in humans (Sumino et al., 1975). Metabolism

of cadmium is limited to binding with low molecular weight proteins (EPA, 1987a). Cadmium tends to accumulate in the kidney and elimination occurs slowly through the urine (EPA, 1984).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

The toxicology of cadmium has been reviewed by Friberg et al. (1974), IARC (1976), EPA (1980, 1981, 1985b,c) and ATSDR (1987). The following sections provide a brief summary of the toxicological information.

CARCINOGENIC POTENTIAL

A relationship between exposure to cadmium and cancer has been suggested by several epidemiological studies. An increased lung cancer rate was observed in workers in a cadmium processing plant (Thun et al., 1985). EPA (1985b) concluded that the increase reported in this study was likely to be due to cadmium exposure and that it could not be explained by smoking or previous occupational exposure to arsenic among the workers. Other epidemiological studies suggest that cadmium may induce lung, prostate and kidney tumors, but EPA (1985b) did not consider these tests conclusive because, in the majority of cases, workers were also exposed to other potentially carcinogenic compounds (EPA, 1985b).

Cadmium has been observed to cause significantly increased tumor incidences in laboratory animals. Inhalation of cadmium chloride has been shown to produce increased incidences of lung carcinomas in rats (Takenaka et al., 1983). Intratracheal administration of cadmium oxide produced an increased incidence of mammary tumors, but not lung tumors, in male rats (Sanders and Mahaffey, 1984). Similar treatment with cadmium chloride produced a 5% incidence of invasive prostatic carcinomas in rats (Hoffmann et al., 1985). Injection of cadmium into laboratory animals results in injection-site sarcomas and testicular tumors of the Leydig cells (EPA, 1981).

Cadmium has not been shown to be carcinogenic in humans or animals by the oral route of administration (ATSDR, 1987).

GENOTOXIC POTENTIAL

ATSDR (1987) has recently reviewed the genotoxic potential of cadmium. In vitro tests for point mutations in bacteria and yeast have been inconclusive, and in vitro and in vivo assays for chromosome aberrations have produced mixed results. However, cadmium has been reported to be mutagenic in an in vitro assay, in mammalian cells (Castro, 1976), and in mouse lymphoma cells (Amacher and Paillet, 1980; Oberly et al., 1982).

REPRODUCTIVE EFFECTS

High parenteral doses of cadmium may cause severe injury to the gonads, especially in males, and this may lead to reduced fertility and sterility. Decreased testosterone levels and depressed sperm viability were reported in male rats injected with 1.8 mg/kg cadmium chloride (Laskey et al., 1984). Cadmium is also teratogenic in animals after injection. Intravenous injection of 2 mg/kg cadmium sulfate produced malformations of the face, limbs and skeleton in hamsters (Gale and Ferm, 1973), and subcutaneous injection of 0.63 mg/kg was reported to be teratogenic in mice (Ishizu et al., 1973).

The majority of chronic oral exposure studies do not reveal significant effects on the reproductive system in adult male or female rats (ATSDR, 1987); however, a few studies have reported general breeding failure following oral administration (Schroeder and Mitchener, 1971; Wills et al., 1981). Oral or inhalation exposure to cadmium compounds has also been reported to produce fetotoxic and developmental effects in the offspring of treated animals, but generally not teratogenic effects. Decreased fetal weight has been observed in the offspring of rats exposed to 200 ppm cadmium chloride in the diet (Pond and Walker, 1975) or airborne concentrations of 0.2-0.6 mg/m^3 (Prigge, 1978); no teratogenic effects were reported in these studies. An increased incidence of resorptions, decreased fetal body weight and retarded skeletal development

was observed in the offspring of rats exposed to 10 mg cadmium/kg/day (as cadmium chloride) in the diet for 6 weeks prior to mating and during gestation (Sutou et al., 1980).

ACUTE/CHRONIC EFFECTS

Acute oral exposure to high cadmium doses leads to gastrointestinal symptoms including nausea, vomiting, abdominal pain, cramps and diarrhea (Bernard and Lauwerys, 1984). Chronic oral exposure to lower doses may produce adverse effects in the kidney, liver, bone, immune system and cardiovascular system. These effects depend on the cadmium concentration in the respective target tissues, and because cadmium is highly retained in the body, similar effects may occur after short-term exposure to high doses or longer-term exposure to low doses (Wang and Foulkes, 1984).

The kidney is the primary target organ for cadmium. Renal tubular dysfunction, and subsequent proteinuria, occur at an increased incidence in humans with renal cortical concentrations of about 200 μg/g wet weight (Friberg et al., 1974). These effects are not readily reversible (Piscator, 1984; Elinder et al., 1985), but they are rarely associated with increased mortality (Friberg et al., 1985). Renal dysfunction may be the primary defect responsible for bone disorders (osteomalacia and osteoporosis) that have also been associated with cadmium exposure in humans (Nomiyama, 1986).

Chronic oral exposure to cadmium has been reported to result in hepatic structural changes (Stowe et al., 1972), hypertension (Kopp, 1986) and alterations in the immune system (Exon and Koller, 1986) in laboratory animals. Parenteral injection of large doses has been associated with testicular (Parizck, 1957) and ovarian (Parizck et al., 1968) damage in rats. Oral doses of 100 mg/kg or higher have also been observed to cause testicular damage (Kotsonis and Klaasen, 1977). Testicular damage has not been reported in occupationally exposed humans (Smith et al., 1960).

Inhalation exposure to high concentrations of cadmium may result in pulmonary irritation (Friberg et al., 1974), pneumonitis and pulmonary edema (Bernard and Lauwerys, 1986). Acute exposure to 0.2-0.5 mg/m^3 has been reported to produce mild, reversible respiratory symptoms characteristic of metal fume fever (Bernard and Lauwerys, 1986). Chronic exposure to lower concentrations may result in chronic obstructive lung disease or renal injury. Evidence of kidney damage has been reported in workers exposed to 20 $\mu g/m^3$ cadmium dust for 27 years (Materne et al., 1975) and in workers exposed to 50 mg/m^3 for 9-12 years (Kjellstrom et al., 1977).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

EPA (1981) has reviewed the health risks posed by airborne cadmium compounds in considerable detail. The International Agency for Research on Cancer (IARC, 1976) has also reviewed the carcinogenicity of cadmium compounds. Both reviews were updated by EPA (1985b), which also presented a risk assessment for exposure to airborne cadmium.

Applying the criteria described in EPA's Guidelines for Assessment of Carcinogenic Risk (EPA, 1986), inhaled cadmium has been classified by EPA (1985a) in Group B1—Probable Human Carcinogen. This category applies to agents for which there is limited evidence of carcinogenicity from human studies and sufficient evidence from animal studies (EPA, 1984).

EPA (1985b) based its quantitative risk assessment for inhaled cadmium on an occupational exposure study reported by Thun et al. (1985). In this study, information on exposure levels and duration were used to compile a single average measure of cumulative worker exposure; the extent to which these exposure estimates deviate from the actual exposure is unknown. The cumulative worker exposure was converted to a lifetime average exposure level for the risk calculations, which assumes that exposure to cadmium at any time during life would lead to the same increase in lifetime risk of lung cancer. The observed lung cancer incidence and the lifetime average exposure level were used to estimate a "unit risk" of 1.8×10^{-3} $(\mu g/m^3)^{-1}$, corresponding to a

cancer potency factor of 6.1 $(mg/kg/day)^{-1}$. However, because of uncertainties in the estimates of both exposure levels and lung cancer response, the range of uncertainties in this unit risk estimate is very wide, ranging from 4.3×10^{-6} to 3.8×10^{-2} $(\mu g/m^3)^{-1}$.

The estimate of unit risk is uncertain and likely to be conservative when applied to the general population. The study on which the estimate is based is not regarded as fully conclusive in showing any increased cancer risk and no dose-response relationship was demonstrated. In addition, the estimated exposure levels are uncertain and the assumption of a linear dose-response relationship is arbitrary.

EPA (1985b) also calculated a unit risk estimate for inhalation of cadmium on the basis of a study in which male Wistar rats were exposed to cadmium chloride aerosols 23 hours/day for 18 months at concentrations of 0, 12.5, 25, and 50 $\mu g/m^3$ (Takenaka et al., 1983). The incidence of lung carcinomas was 0/38, 6/39, 20/38, and 25/35 in the four treatment groups. EPA derived a unit risk of 5.5×10^{-2} $(\mu g/m^3)^{-1}$ from these data. Although this estimate is higher than the estimate made on the basis of the epidemiological data of Thun et al. (1985), EPA (1985b) concluded that the latter is more reliable because the types of cadmium compounds to which humans are exposed differ from the cadmium chloride used in the rat study and because interspecies differences do not have to be taken into consideration.

The maximum contaminant level (MCL) established for cadmium by EPA in its national interim primary drinking water standards and the Ambient Water Quality Criterion for the protection of human health are both 10 μg/liter (EPA, 1980; CFR, 1984). EPA established the standard for cadmium on the basis of the "generally accepted" estimate of 200 $\mu g/g$ wet weight of cadmium in the renal cortex as the critical concentration for renal toxicity. Friberg et al. (1974) estimated that daily ingestion of 250-350 μg cadmium over 50 years would result in such renal concentrations. Other more recent reviews suggest that 200 μg/day is an acceptable daily limit for cadmium intake.

EPA has also proposed a maximum contaminant level goal (MCLG) of 5 μg/liter. The proposed MCLG is equal to the NAS and WHO guidelines of 5 μg/liter (EPA, 1985a).

EPA (1985b) derived two different RfDs for cadmium using renal toxicity as the toxic endpoint and a safety factor of 10. The RfD associated with oral exposure to cadmium in drinking water is 5×10^{-4} mg/kg/day, based upon the LOAEL of 5×10^{-3} mg/kg identified in humans. EPA also derived an RfD of 1×10^{-3} mg/kg/day for oral exposure to cadmium in food or other nonaqueous exposure media.

The American Conference of Governmental Industrial Hygienists (ACGIH, 1986) recommends a time-weighted average Threshold Limit Value (TLV) of 0.05 mg/m^3 for all forms of cadmium dusts and salts and a ceiling limit of 0.05 mg/m^3 for cadmium oxide fume.

SUMMARY OF CADMIUM CRITERIA

EPA carcinogen classification (inhalation only)	Group B1
Inhalation carcinogenic potency factor	6.1 $(mg/kg/day)^{-1}$
RfD	
Drinking Water Exposure	5×10^{-4} mg/kg/day
Other Oral Routes	1×10^{-3} mg/kg/day
MCL	10 μg/liter
Proposed MCLG	5 μg/liter

REFERENCES

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987. Draft Toxicological Profile for Cadmium. U.S. Public Health Service. November 1987.

AMACHER, D.E. and PAILLET, S.C. 1980. Induction of trifluorothymidine-resistant mutants by metal ions in L51784/$TK^{+}/^{-}$ cells. Mutat. Res 78:279-288 (as cited in ATSDR, 1987)

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th Ed. Cincinnati, Ohio

AUGHEY, E., FELL, G.S., SCOTT, R., and BLACK, M. 1984. Histopathology of early effects of oral cadmium in the rat kidney. Environ. Health Perspect. 54:153-161

BERNARD, A. and LAUWERYS, R. 1984. Cadmium in human population. Experimentia 40:143-152

BERNARD, A. and LAUWERYS, R. 1986. Effects of cadmium exposure in humans. In: Foulkes EC (ed). Handbook of experimental pharmacology, Vol. 80. Berlin: Springer Verlag, pp. 135-177 (as cited in ATSDR, 1987)

CASTRO, B. 1976. Letter to Richard Trost, Environmental Protection Agency, enclosing mutagenicity data on cadmium chloride and cadmium acetate (as cite in ATSDR 1987).

CODE OF FEDERAL REGULATIONS (CFR). 1984. 40 CFR 141, Environmental Protection Agency, National Interim Primary Drinking Water Regulations. U.S. Government Printing Office, Washington, D.C.

ELINDER, C.G, EDLING, C., LINDBERG, E., KAGEDAL, B., and VESTERBORG, O. 1985. Assessment of renal function in workers previously exposed to cadmium. Brit. J. Ind. Med. 42: 754-760 (as cited in ATSDR, 1987)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Washington, D.C. December 1979. EPA 440/4-79-029

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Cadmium. Environmental Criteria and Assessment Office. Cincinnati, Ohio. EPA 440/5-80-025

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1981. Health Effects Document for Cadmium. Research Triangle Park, North Carolina. EPA 600/8-81-023.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Cadmium. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-038

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. National primary drinking water regulations; synthetic organic chemicals, inorganic chemicals and microorganisms; proposed rule. 40 CFR Part 141. Fed. Reg. 50(219):46936-47025 (November 13, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. Updated Mutagenicity and Carcinogenicity Assessment of Cadmium. Addendum to the Health Assessment Document for Cadmium (May 1981; EPA 600/8-81/023). Office of Health and Environmental Assessment, Washington, D.C. June 1985. EPA 600/8-83-025F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985c. Drinking Water Criteria Document for Cadmium (Final Draft). Office of Drinking Water, Washington, D.C. April 1985. PB86 117934/AS

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Guidelines for carcinogenic risk assessment. Fed. Reg. 51: 33992-34003. (September 24, 1986)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987a. Health Advisory for Cadmium. Office of Drinking Water, Washington, D.C. March 31, 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987b. Integrated Risk Information System (IRIS). Reference dose (RfD): description and use in health risk assessments. Appendix A (as cited in ATSDR, 1987)

EXON, J.H. and KOLLER, L.D. 1986. Immunotoxicity of cadmium. In: Foulkes EC (ed). Handbook of experimental pharmacology Vol. 80. Berlin: Springer Verlag, pp. 341-350. (as cited in ATSDR, 1987)

FRIBERG, L., ELINDER, C.G., KJELLSTROM, T., and NORDBERG, G.F. 1985. Cadmium and Health: a toxicological and epidemiological appraisal. Boca Raton, FL. CRC Press (as cited in ATSDR, 1987)

FRIBERG, L.T., PISCATOR, M., and NORDBERG, G. 1974. Cadmium in the Environment. 2nd ed. CRC Press, Cleveland, Ohio

FROST, R.R. and GRIFFIN, R.A. 1977. Effect of pH on adsorption of copper, zinc, and cadmium from landfill leachate by clay minerals. J. Environ. Sci. Health 12:138-156

GALE, T.F. and FERM, V.H. 1973. Skeletal malformations resulting from cadmium treastment in the hamster. Biol. Neonate 19:149-160. (as cited in ATSDR, 1987)

GARDINER, J. 1974. The chemistry of cadmium in natural waters – II. The adsorption of cadmium on river muds and naturally occurring solids. Water Research. 8:157-164

HOFFMAN, L., PUTZKE, H.P., KAMPEHL, H.J., RUSSBULT, R., GASE, P., SIMONN, C., ERDMANN, T., and HUCKSTORF, C. 1985. Carcinogenic effects of cadmium on the prostate of the rat. J. Cancer Res. Clin. Oncol. 109:193-199. (as cited in ATSDR, 1987)

HUANG, C.P., ELLIOTT, H.A., and ASHMEAD, R.M. 1977. Interfacial reactions and the fate of heavy metals in soil-water system. J. Water Pollut. Control Fed. 49(5):745-756

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1976. IARC Monographs on the Carcinogenic Risk of Chemicals to Man. Vol. 11: Cadmium, Nickel, Some Epoxides, Miscellaneous Industrial Chemicals, and General Considerations on Volatile Anesthetics. World Health Organization, Lyon, France. pp. 39-74

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1982. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol. 29: Some Industrial Chemicals and Dyestuffs. World Health Organization, Lyon, France

ISHIZU, S., MINAMI, M., SUZUKI, A., YAMADA, M., SATO, M., and YAMAMURA, K. 1973. An experimental study on teratogenic effects of cadmium. Ind. Health 11:127:139. (as cited in ATSDR, 1987)

KABATA-PENDIAS, A. and PENDIAS, H. 1984. Trace Elements in Soils and Plants. CRC Press, Boca Raton

KJELLSTROM, T., EVRIN, P.E., and RAHNSTE, B. 1977. Dose-response analysis of cadmium-induced tubular proteinuria. A study of urinary B_2-microglobulin excretion among workers in a battery factory. Environ. Res. 13:303-317 (as cited in ATSDR, 1987)

KOPP, S.J. 1986. Cadmium and the cardiovascular system. In: Foulkes, E.C. (ed). Handbook of experimental pharmacology. Vol. 80. Berlin: Springer Verlag, pp. 195-280. (as cited in ATSDR, 1987)

KORTE, N.E., SKOPP, J., FULLER, W.H., NIEVLA, E.E., and ALESII, B.A. 1976. Trace element movement in soils: Influence of soil physical and chemical properties. Soil Sci. 122:350-525

KOSTIAL, K., KELLO, D., JUGO, S., RABAR, I., and MALJKOVIC, T. 1978. Influence of age on metal metabolism and toxicity. Environ. Health Perspect. 25:81-86. (as cited in ATSDR, 1987)

KOTSONIS, F.N. and KLAASSEN, C.D. 1977. Toxicity and distribution of cadmium administered to rats at sublethal doses. Toxicol. Appl. Pharmacol. 73:250-255. (as cited in ATSDR, 1987)

LASKEY, J.W., REHNBERG, G.L., LAWS, S.C., and HEIN, J.F., 1984. Reproductive effects of low acute doses of cadmium chloride in adult male rats. Toxicol. Appl. Pharmacol. 73:250-255. (as cited in ATSDR, 1987)

MATERNE, D., LAUWERYS, R., BUCHET, J.P., ROELS, H., BROUWERS, J., and STANESCU, D. 1975. Investigations sur le risques resultant de l'exposition au cadmium dans deux enterprises de production et deux entreprises d'utilisation du cadmium. Cahiers de Medecine du Travail XII:1. (as cited in ATSDR, 1987)

MERANGER, J.C., SUBRAMIAN, K.S., and CHALIFOUX, C. 1981. Survey for cadmium, cobalt, chromium, copper, nickel, lead, zinc, calcium and magnesium in Canadian drinking water supplies. J. Assoc. Off. Anal. Chem. 64:44. (as cited in ATSDR, 1987)

MOORE, J.W. and RAMAMOORTHY, S. 1984. Heavy Metals in Natural Waters. Springer-Verlag, New York

NOMIYAMA, K. 1986. The chronic toxicity of cadmium. In: Foulkes, E.C. (ed). Handbook of experimental pharmacology. Vol. 80. Berlin: SpringerVerlag, pp. 101-133. (as cited in ATSDR, 1987)

OBERLY, T.J., PIPER, C.E., and McDONALD, D.S. 1982. Mutagenicity of metal salts in the L5178 Y mouse lymphoma assay. J. Toxicol. Environ. Health 9:367:376. (as cited in ATSDR, 1987)

PARIZEK, J. 1957. The destructive effect of cadmium ion on testicular tissue and its prevention by zinc. J. Endocrinol. 15:56-63. (as cited in ATSDR, 1987)

PARIZEK, J., OSTADALOVA, I., BENES, I., and BABICK, A. 1968. The effect of a subcutaneous injection of cadmium salts on the ovaries of adult rats in persistent oestrus. J. Repro. Fertil. 17:559:562. (as cited in ATSDR, 1987)

PISCATOR, M., 1984. Long-term observations on tubular and glomerular function in cadmium-exposed persons. Environ. Health Perspect. 54:175-179. (as cited in ATSDR, 1987)

POND, W.G. and WALKER, E.F. 1975. Effect of dietary Ca and Cd level of pregnant rats on reproduction and on dam and progeny tissue mineral concentrations. Proc. Soc. Exp. Biol. Med. 148:655. (as cited in ATSDR, 1987)

PRIGGE, E. 1978. Inhalative cadmium effects in pregnant and fetal rats. Toxicol. 10:297-309.

SABBIONI, E., MARAFANTE, E., AMANTINI, L., UBERTALLI, L., and PIETRA, R. 1978. (As cited in EPA Health Advisory for Cadmium, 1987) Sci. Total. Environ. 10:135

SANDERS, C.L. and MAHAFFEY, J.A. 1984. Carcinogenicity of single and multiple intratracheal instillations of cadmium oxide in the rat. Environ. Res. 33:227-233.

SASSER, L.B. and JARBOE, G.E. 1977. Intestinal absorption and retention of cadmium in neonatal rat. Toxicol. Appl. Pharmacol. 41:423:431. (as cited in ATSDR, 1987)

SCHROEDER, H.A. and MITCHNER, M. 1971. Toxic effects of trace elements on the reproduction of mice and rats. Arch. Environ. Health 12:81-96. (as cited in ATSDR, 1987)

SMITH, J.P., SMITH, J.C., and McCALL, A.J. 1960. Chronic poisoning from cadmium fume. J. Pathol. Bacteriol. 80:287-297. (as cited in ATSDR, 1987)

STOWE, H.D., WILSON, M., and GOYER, R.A. 1972. Clinical and morphological effects of oral cadmium toxicity in rabbits. Arch. Pathol. 94:389-405.

SUMINO, K., HAYAKAWA, K., SHIBATA, T., and KITAMURA, S. 1975a. Heavy metals in normal Japanese tissues. Arch. Environ. Health 30:487-494. (As cited in EPA, 1987)

SUTOU, S., YAMAMOTO, K., SENDOTA, H., and SUGIYAMA, M. 1980. Toxicity, fertility, teratogenicity and dominant lethal test in rats and administered cadmium subchronically. III. Fertility, teratogenicity, and dominant lethal test. Ecotoxicol. Environ. Safety 4:51-56.

TAKENAKA, S., OLDIGES, H., KONIG, H., HOCHRAINER, D., and OBERDORSTER, G. 1983. Carcinogenicity of cadmium chloride aerosols in Wistar rats. J. Natl. Cancer Inst. 70:367:373.

THUN, M.J., SCHNORR, T.M., SMITH, A.B., HALPERIN, W.E., and LEMEN, B.A. 1985. Mortality among a cohort of U.S. Cadmium production workers - An update. J. Natl. Cancer Inst. 74:325-333

WAHLBERG, J.E. 1965. Percutaneous toxicity of metal compounds. Arch. Environ. Health 11:201-204. (as cited in ATSDR, 1987)

WANG, X.P. and FOULKES, E.C. 1984. Specificity of acute effects of cadmium on renal function. Toxicol. 30:243-247. (as cited in ATSDR, 1987)

WILLS, J.H., GROBLEWSKI, G.E., and COULSTON, F. 1981. Chronic and multigeneration toxicities of small concentrations of cadmium in the diet of rats. Ecotox. Environ. Safety 5:452-464. (as cited in ATSDR, 1987).

A.4 CHLOROFORM

INTRODUCTION

Chloroform is a dense, colorless, volatile organic chemical. It is used as a raw material in the chemical industry for the manufacture of fluorocarbons, pesticides and dyes, and as an extractant and industrial solvent. In the past, chloroform was used as an anesthetic (EPA, 1985). U.S. ambient air concentrations have been estimated to be in the ranges of 0.02-0.2 $\mu g/m^3$ in rural areas and 0.2-3.4 $\mu g/m^3$ in urban areas (ATSDR, 1987). Concentrations identified in U.S. national drinking water surveys generally fall within the range of 32-68 μg/liter with a maximum concentration of 311 μg/liter identified (Brass et al., 1977; Symons et al., 1975). In other studies, concentrations as high as 0.37 μg/liter have been identified in surface water (Strachar and Edwards, 1984) and 490 μg/liter in groundwater (Ras et al., 1985).

TOXICOKINETICS

Chloroform is rapidly absorbed through the respiratory tract and gastrointestinal tract. Dermal absorption from contact of the skin with liquid chloroform can also occur. The extent of absorption depends on the fat content of the individual. Following inhalation or ingestion, chloroform is found at high concentrations in the adipose tissue, brain, liver and kidneys. The liver is the primary site for chloroform metabolism. Elimination is primarily through exhalation of the unmetabolized compound and the major end-product of chloroform metabolism, carbon dioxide. The inorganic chlorine generated from chloroform metabolism is excreted through the urine (EPA, 1985).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

CARCINOGENIC POTENTIAL

Although no epidemiological studies have evaluated the carcinogenicity of chloroform itself, several have focused on populations exposed to chlorinated drinking water in which chloroform is the predominant chlorinated hydrocarbon present. Small statistically significant increases in rectal, bladder, and colon cancers have been observed in several studies; however, because other potential carcinogens were present along with chloroform, EPA (1985) concluded that it is impossible to determine whether chloroform itself causes cancer in humans.

The carcinogenicity of chloroform has been extensively studied in experimental animals. Chloroform has been reported to induce renal epithelial tumors in male Osborne-Mendel rats (Jorgenson et al., 1985; NCI, 1976), hepatocellular carcinomas in male and female $B6C3F_1$ mice (NCI, 1976), and kidney tumors in male ICI mice (Roe et al., 1979).

GENOTOXIC POTENTIAL

The genotoxicity of chloroform has been examined in a number of *in vitro* assays including tests for point mutations in bacteria, yeast, and mammalian cells, DNA damage in yeast and mammalian cells and chromosomal effects. These studies have been reviewed by EPA (1985) and ATSDR (1987). Bacterial tests with chloroform have been predominantly negative. However, EPA (1985) considers many of the results to be inconclusive because of inadequacies in the experimental protocols, including inadequate evidence that chloroform was activated or metabolized by the activation system used and the possibility that the presumed genotoxic metabolite of chloroform, phosgene, may not have reached the bacterial DNA. Results in eukaryotic test systems were also primarily negative and EPA (1985) considered these studies also to be inconclusive. Results of *in vivo* assays for gene mutations and DNA damage

were negative while the results of tests for chromosome abnormalities were mixed.

REPRODUCTIVE EFFECTS

There is no information available on the reproductive toxicity of chloroform in humans. In experimental animals, a number of studies indicate that chloroform can cause adverse effects in pregnancy maintenance, delays in fetal development, and fetal abnormalities in experimental animals (EPA, 1985). Schwetz et al. (1974) exposed female Sprague-Dawley rats via inhalation 7 hours per day to 30, 100, or 300 ppm reagent-grade chloroform on days 6-15 of gestation. Fetotoxicity and retarded development were noted at all dose levels; maternal toxicity was reported at the 100 and 300 ppm dose levels. The investigators reported a significant increase in the number of fetal resorptions at 300 ppm and an apparent decrease in conception rate. At 100 ppm, incidences of acaudia (absence of tail), short tail, imperforate anus, subcutaneous edema, missing ribs, and delayed ossification of sternebrae were increased. Fetotoxic effects were also noted in mice exposed via inhalation to 100 ppm (Murray et al., 1979). Oral exposure has been reported to result in increased numbers of resorptions and decreased fetal body weights (but only at maternally toxic levels) in rats (Thompson et al., 1974), decreased neo-natal body weight gains in mice (Burkhalter and Balster, 1979), and decreased fetal body weights and retarded skeletal development in rabbits (Thompson et al., 1974).

Studies indicate that exposure to chloroform may be associated with adverse effects on reproductive organs in adult animals. Sperm abnormalities have been reported in male mice exposed via inhalation 4 hours/day for 5 days to 400 or 800 ppm doses (Land et al., 1981) and gonadal atrophy has been reported in male and female rats exposed by gavage to doses of 410 mg/kg/day 6 days/week for 13 weeks in a toothpaste vehicle (Palmer et al., 1979).

ACUTE/CHRONIC EFFECTS

The toxicity of chloroform has been reviewed by EPA (1985) and ATSDR (1987). Chloroform exposure affects primarily the liver, kidney, and central nervous system.

In humans, acute exposure to very high doses may result in rapid death due to ventricular fibrillation (caused by cardiac sensitization to epinephrine) or in delayed death due to liver and kidney damage. In the past, human exposure to potentially fatal doses was often the result of chloroform's use as an anaesthetic gas. Dose levels necessary for anaesthesia (20,000 - 40,000 ppm in air) are extremely high (EPA, 1985). Other effects that may result from acute exposure include irritation of the skin, eyes and gastrointestinal tract. Chronic exposure to airborne chloroform has been associated with hepatotoxic effects in workers exposed to 2-205 ppm (Bomski et al., 1967), or 14->400 ppm (Phoon et al., 1983). However, lack of appropriate control groups in these studies limits the conclusions that can be drawn from these reports (EPA, 1985). Chronic oral ingestion of 1.6 - 2.6 g/day chloroform has also been associated with hepatic and renal toxicity (Wallace, 1950).

In experimental animals, narcosis has been reported to result from acute inhalation exposure to 2500 ppm in mice (EPA, 1985) and a single oral dose of 350 mg/kg in rats (Jones et al., 1958). Exposure to lower doses results in less severe symptoms of central nervous system depression as well as hepatic and renal histopathological alterations. Torkelson et al. (1976) exposed several species via inhalation to 25, 50 or 85 ppm chloroform for 7 hours/day 5 days/week for 6 months or to 25 ppm 1-4 hours/day for 6 months. Exposure to 25 ppm for up to 4 hours/day produced no adverse effects in rats. Exposure to 25 ppm chloroform for 7 hours/day resulted in lobular granular degeneration and focal necrosis of the liver and cloudy swelling of the kidneys in rats, guinea pigs, and rabbits. More severe changes were seen in rats exposed to 50 or 85 ppm for 7 hours/day but not in guinea pigs or rabbits. Roe et al. (1979) observed severe renal changes and an increased incidence of renal

tumors, but no liver effects, in mice exposed by gavage to 60 mg/kg/day chloroform, 6 days/week for 80 weeks. This study also reported mouse strain and sex differences in susceptibility to renal effects of chloroform.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

Based on EPA's proposed guidelines for carcinogen risk assessment (EPA, 1984b), chloroform has been classified in Group B2—Probable Human Carcinogen (EPA, 1985). This category applies to agents for which there is sufficient animal evidence and inadequate human evidence of carcinogenicity.

In its Health Assessment Document for chloroform, EPA (1985) reported carcinogenic potency factors for exposure by inhalation or ingestion. These values were based on the results of a National Cancer Institute bioassay in mice (NCI, 1976). Groups of 50 male and 50 female $B6C3F_1$ mice were treated by gavage 5 times/week for 78 weeks with chloroform in corn oil. The male and female mice received average doses of 138 or 277 mg/kg and 238 or 477 mg/kg, respectively, 5 times/week for 78 weeks. The mice were sacrificed after 92 or 93 weeks. The incidence of hepatocellular carcinomas was significantly increased in both male (1/18, 18/50, 44/50) and female mice (0/20, 36/45, 39/41) (control, low dose, and high dose, respectively, for both sexes). A carcinogenic potency factor for inhalation exposure of 8.1×10^{-2} $(mg/kg/day)^{-1}$ was derived by taking the geometric mean of two estimates calculated for male and female mice individually. This potency factor is a plausible 95% upper limit; the true value is not likely to be higher, but may be lower than this estimate. This value has been verified by EPA and is considered appropriate for use in estimating possible risks associated with inhalation exposure (ATSDR, 1987).

EPA (1987) derived a carcinogenic potency factor for oral exposure to chloroform based on the induction of kidney tumors in male rats reported by Jorgenson et al. (1985). In this study, male rats and female mice received time-weighted average doses of 0, 19, 38, 81 and 160 mg/kg/day and 0, 34, 65, 130 and 263 mg/kg/day, respectively for 104 weeks. Renal tubular cell

adenomas and/or adenocarcinomas were found in male rats at incidences of 4/301, 4/313, 4/148, 3/48, and 7/50 in the 0, 19, 38, 81 and 160 mg/kg/day dose groups, respectively. There were no treatment-related effects in mice. EPA (1987) calculated a cancer potency factor of 6.1×10^{-3} $(mg/kg/day)^{-1}$ based on the renal tumor data in rats. This estimated potency factor is a plausible 95% upper limit; the true value in not likely to be higher, but may be lower than this estimate.

EPA derived a reference dose (RfD) for oral exposure to chloroform (EPA, 1986) based on a chronic oral bioassay in dogs (Heywood et al., 1979). In this study, beagle dogs were administered chloroform at doses of 15 or 30 mg/kg/day in a toothpaste base for 6 days/week for 7.5 years. Fatty cysts were observed in the livers of some animals in the treatment groups. Nodules of altered hepatocytes were observed that appeared to be treatment-related, but not dose-dependent. The 15 mg/kg/day dose was considered to be the lowest-observed-adverse effect level (LOAEL), and an RfD of 0.01 mg/kg/day was calculated.

The safe drinking water maximum contaminant level (MCL) for total trihalomethanes is 100 μg/liter. Under this regulation, chloroform is one of four trihalomethanes whose combined concentrations must not exceed this value. In addition to health effects, other factors, such as technical feasibility and costs, are considered in developing MCLs (EPA, 1978).

The American Conference of Governmental Industrial Hygienists (ACGIH, 1986) recommended a time-weighted average threshold limit value of 10 ppm (50 mg/m^3) for occupational exposure to chloroform.

SUMMARY OF CHLOROFORM CRITERIA

EPA carcinogen classification	Group B2
Oral Carcinogenic potency factor	6.1×10^{-3} $(mg/kg/day)^{-1}$
Inhalation Carcinogenic potency factor	8.1×10^{-2} $(mg/kg/day)^{-1}$
RfD, verified	0.01 mg/kg/day
MCL (total trihalomethanes)	100 μg/liter
MCLG	Not available

REFERENCES

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987. Draft Toxicological Profile for Chloroform. U.S. Public Health Service.

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of Threshold Limit Values and Biological Exposure Indices. 5th ed. ACGIH, Cincinnati, Ohio

BOMSKI, H., SOBOLWESKA, A., and STRAKOWSKI, A. 1967. Toxic damage of the liver by chloroform in chemical industry workers. Arch. Gewer be pathol. Gewer behy. 24:127-134. (FRG) (As cited in ATSDR, 1987)

BRASS, H.J., FEIGE, M.A., HALLORAN, T., MELLO, J.W., MUNCH, D., and THOMAS, R.F., 1977. The National Organic Monitoring Survey Sampling and Analysis For Purgeable Organic Compounds. (As cited in ATSDR, 1987)

BURKHALTER, J. and BALSTER, R.L. 1979. Behavioral teratology evaluation of chloroform in mice. Neurobehav. Toxicol. 1:199-205 (As cited in ATSDR, 1987)

DILLING, W.L., TEFERTILLER, N.B., and KAOOLS, G.J. 1975. Evaporation rates of methylene chloride, chloroform, 1,1,1-trichloroethane, trichloroethylene, tetrachloroethylene, and other chlorinated compounds in dilute aqueous solutions. Environ. Sci. Technol. 9(9):833-838 (As cited in EPA, 1979)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1978. Statement of Basis and Purpose for an Amendment to the National Interim Primary Drinking Water Regulations on Trihalomethanes. Office of Water Supply, Washington, D.C. (As cited in EPA, 1980)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Washington, D.C. December 1979. EPA 440/4-79-029

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Chloroform. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-033

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984a. Health Effects Assessment for Chloroform. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-010

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984b. Proposed guidelines for carcinogen risk assessment. Fed. Reg. 49:46294-46299 (November 23, 1984)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Health Assessment Document for Chloroform. Environmental Criteria and Assessment Office, Research Triangle Park, North Carolina. September 1985. EPA 600/8-84-004F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986a. Verified Reference Doses of the U.S. EPA (including addendum of May 14, 1986). Prepared by the RfD Work Group of the Risk Assessment Forum, Washington, D.C. January 1986. ECAO-CIN-475

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986b. Superfund Public Health Evaluation Manual. Office of Solid Waste and Emergency Response, Washington, D.C. October 1986. EPA/540/1-86/060

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Integrated Risk Information System (IRIS). Risk estimate for carcinogenicity for chloroform. On line:Input pending. (Verification date 8/26/87). Cincinnati, OH: Office of Health and Environmental Assessment, Environmental Criteria Assessment and Office (As cited in ATSDR, 1987)

HEYWOOD, R., SORTWELL, R.J., NOEL, P.R.B., et al. 1979. Safety evaluation of toothpaste containing chloroform. III. Long-term study in beagle dogs. J. Environ. Pathol. Toxicol. 2:835-851 (As cited in EPA, 1986)

JONES, W.M., MARGOLIS, G., and STEPHEN, C.R. 1958. Hepatotoxicity of inhalation anesthetic drugs, Anesthesiology. 19:715.

JORGENSON, T.A., MEIERHENRY, E.F., RUSHBROOK, C.J., et al. 1985. Carcinogenicity of chloroform in drinking water to male Osborne-Mendel rats and female B6C3F_1 mice. Fund. Appl. Toxicol. (U.S.A.); 5(4): 760-769. (As cited in ATSDR, 1987)

LAND, P.D., OWEN, E.L., and LINDE, H.W. 1981. Morphologic changes in mouse spermatozoa after exposure to inhalational anesthetics during early spermatogenesis. Anesthesiology; 54:53-56 (As cited in ATSDR, 1987)

MURRAY, F.J., SCHWETZ, B.A., MCBRIDE, J.G., and STAPLES, R.E. 1979. Toxicity of inhaled chloroform in pregnant mice and their offspring. Toxicol. Appl. Pharmacol.; 50:525-522 (As cite in ATSDR 1987)

NATIONAL CANCER INSTITUTE (NCI). 1976. Carcinogenesis Bioassay of Chloroform. CAS No. 67-66-3. NCI Carcinogenesis Technical Report Series No. 0. Bethesda, Md. DHEW (NIH) Publication No. 76

NATIONAL CANCER INSTITUTE (NCI). 1976. Report on carcinogenesis bioassay of chloroform. NTIS PB-264018. (As cited in ATSDR, 1987)

PALMER, A.K., STREET, A.E., ROE, F.J.C., WORDEN, A.W., and VAN ABBE, N.J. 1979. Safety evaluation of toothpaste containing chloroform. II. Long-term studies in rats. J. Environ. Pathol. Toxicol.; 2:821-833 (As cited in ATSDR, 1987)

PEARSON, C.R. and McCONNELL, G. 1975. Chlorinated C_1 and C_2 hydrocarbons in the marine environment. Prog. R. Soc. Lond. B. 189:305:332 (As cited in EPA, 1979)

PHOON, W.A., GOH, K.T., LEE, L.T., TAN, K.T., and KWOK, S.E. 1983. Toxic Jaundice from occupational exposure to chloroform. Med. J. Malaysia; 30:31-34. (As cited in ATSDR, 1987)

RAO, P.S.C., HORNSBY, A.G., and JESSUP, R.E. 1985. Indices for ranking the potential for pesticide contamination of groundwater. Soil Crop Sci. Soc. Fl Proc. 44:1-8 (as cited in ATSDR, 1987).

ROE, F.J.C., PALMER, A.A.K., WORDEN, A.N., and VAN ABBE, N.J. 1979. Safety evaluation of toothpaste containing chloroform. I. Long-term studies in mice. J. Environ. Toxicol.; 2:799-819. (As cited in ATSDR, 1987)

SCHWETZ, B.A., LEONG, B.K.J., and GEHRING, P.J. 1974. Embryo- and fetotoxicity of inhaled chloroform in rats. Toxicol. Appl. Pharmacol. 28:442-451 (As cited in EPA, 1985)

STRACHAN, W.M.J. and EDWARDS, C.J. 1984. Organic pollutants in Lake Ontario. Adv. Environ. Sci. Technol. 14:239-264 (as cited in ATSDR, 1987).

SYMONS, J.M., BELLAR, T.A., CARSWELL, J.K., et al. 1975. National Organic Reconnaisance Survey for Halogenated Organics. J. Am. Water Works Assoc.; 67:634 (As cited in ATSDR, 1987)

THOMPSON, D.J., WATNET, S.D., and ROBINSON, V.V. 1974. Teratology studies on orally administered chloroform in the rat and rabbit. Toxicol. Appl. Pharmacol.; 29:348-357. (As cited in ATSDR, 1987)

TORKELSON, T.R., OYEN, F., and ROWE, V.K. 1976. The toxicity of chloroform as determined by single and repeated exposure of laboratory animals. Am. Ind. Hyg. Assoc. J.; 37:697-705 (As cited in EPA, 1985)

VERSCHUEREN, K. 1983. Handbook of Environmental Data on Organic Chemicals. Van Nostrand Reinhold Co., New York

WALLACE, C.J. 1950. Hepatitis and nephrosis due to cough syrup containing chloroform. Calif. Med. 73:442 (as cited in ATSDR, 1987).

WEAST, R.E., ed. 1983. Handbook of Chemistry and Physics. 63rd ed. CRC Press, Cleveland, Ohio.

WILSON, J.T., ENFIELD, C.G., DUNLAP, W.J., CROSBY, R.L., FOSTER, D.A., and BASKIN, L.B. 1981. Transport and fate of selected organic pollutants in a sandy soil. J. Environ. Qual. 10:501-506.

ZOETMAN, B.C.J., HARMON, K., LINDERS, J.B.H.J., and MURRA, C.F.H. 1980. Persistent organic pollutants in river water and groundwater of the Netherlands. Chemosphere; 9:231-249 (As cited in ATSDR, 1987).

A.5 LEAD

INTRODUCTION

Elemental lead is heavy, ductile, and bluish-white in color. It is used widely in industry because of its softness, resistance to corrosion and radiation, and high density. Lead is also used as a paint pigment, in solders, and in storage batteries.

Ambient air concentrations of lead have been found to range between 0.5-2 $\mu g/m^3$. Air near heavy traffic typically has higher concentrations of lead (5-10 $\mu g/m^3$) (NAS, 1980). Considering that consumption of leaded gasoline has been reduced in recent years, current atmospheric lead concentration in urban air are expected to be lower than the levels indicated by currently available data (EPA, 1986b). Water concentrations range from less than 1 to 20 $\mu g/l$ (NAS, 1980).

TOXICOKINETICS

Absorption of lead from the gastrointestinal tract is estimated at 10-15% (Hammond, 1982; Chamberlain et al., 1978). In children absorption tends to be higher; average absorption of lead has been reported as 41% in infants (Goyer, 1986). Dietary lipids, for example lactose, increase the absorption of lead. For adult humans, the deposition rate of particulate airborne lead in the lungs is 30-50%, and essentially all of the lead deposited is absorbed (EPA, 1986a); lead absorption by lungs is particle-size dependent (Goyer, 1988). A study by Nozaki (1966) reported inverse relationships between respiration rate, particle size and lung deposition. Respiratory uptake by children appears to be greater on a body weight basis than adults (Hofmann, 1982). More than 95% of blood lead in humans is associated with erythrocytes (Goyer, 1988). The remaining 5% is transported in plasma and extracellular fluid to the various body organs. Children under 7 years of age have higher blood lead levels than older children (Goyer, 1986). Lead is stored in the body primarily in bone and teeth (Barry, 1975; Schroeder and Tipton, 1968;

Horiguchi and Utsunomiya, 1973; Horiuchi et al., 1959). Accumulation begins during fetal development (Barltrop, 1969; Horiuchi et al., 1959) and continues until 50-60 years of age. Lead is generally not stored in soft tissues but some accumulation may take place in the kidney cortex (Indrasparit et al., 1974).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Lead has diverse biological effects in humans and animals. Though considerable data exist on the effects of lead exposure in humans, exposure is generally stated in terms of blood lead levels rather than in terms of the estimated environmental exposure or administered dose.

A major problem associated with identifying sources of lead exposure is that it is a natural soil constituent. Thus, substantial exposure to lead may occur in regions where soil has a naturally high lead content. This background exposure must be considered in determining the possible significance of additional sources of exposure.

CARCINOGENIC POTENTIAL

The association of occupational lead exposure and increased cancer rates has been examined in several epidemiological studies; however, EPA (1986b) concluded that available data do not provide a sufficient basis for determining whether lead has a carcinogenic potential in humans. These studies were considered to be limited because the identity of the specific lead compound(s), exposure route(s) and/or the concentrations to which workers were exposed were not reported.

Several lead compounds have been reported to cause increased tumor incidences in laboratory animals. In rats, ingestion of 500-2000 mg/kg lead acetate (Azar et al., 1973) or lead subacetate (Van Esch et al., 1962) and subcutaneous administration of lead phosphate (Zollinger, 1953; Balo et al., 1965) have been associated with increased incidences of renal tumors. In

mice, ingestion of lead subacetate has also been associated with increased renal tumor incidences (Van Esch and Kroes, 1969). There is limited evidence for the carcinogenic potential of lead in humans (Goyer, 1986). A slight excess of deaths from cancer was reported for more than 7,000 workers exposed to lead (Cooper & Gaffey, 1975), though the most common tumors were of respiratory and digestive origin. Baker et al. (1980) and Lilis (1981) have reported cases of renal adenocarcinomas in workers with prolonged exposure to lead.

GENOTOXIC POTENTIAL

EPA (1986b) and ATSDR (1988) have reviewed numerous studies on the genotoxicity of lead compounds. EPA (1986b) concluded that the weight of the evidence suggests that lead has a clastogenic potential. Positive results have been reported in approximately half the tests for chromosome aberrations or sister chromatid exchanges in lymphocytes from humans exposed to lead, and in in vitro human lymphocyte cultures exposed to lead acetate. Lead salts have not been shown to be mutagenic in bacterial or mammalian cell test systems; however, positive results have been obtained in plants.

REPRODUCTIVE EFFECTS

Lead exposure has been associated with spontaneous abortions (Nordstrom et al., 1978, 1979), premature delivery and early membrane rupture in humans (Fahim et al., 1976); however, reliable exposure estimates are lacking in most cases (EPA, 1986b). Decreased fertility (Varma et al., 1974) and fetotoxic effects including small litter size and decreased fetal body weight (Maisin et al., 1975; Schroeder and Mitchener, 1971; Stowe and Goyer, 1971) have been reported in laboratory rodents exposed to lead. Skeletal malformations have been reported in rats, mice and hamsters after lead injection (Leonard et al., 1984). Malformations have not been reported when exposure is by inhalation or ingestion (ATSDR, 1988).

Lead has also been reported to produce adverse effects on the reproductive systems of adult animals. In males, testicular atrophy and cellular degeneration have been observed in rats (Chowdhury et al., 1984; Hilderbrand et al., 1973). Morphological sperm abnormalities (Bruce and Heddle, 1979; Maisin et al., 1975; Stowe et al., 1973) and declines in sperm motility (Krasovskii et al., 1979) have also been reported. In females, lead has been associated with decreased ovarian weight (Der et al., 1974) and increased numbers of ovarian cysts (Hilderbrand et al., 1973). In a study by McMichael et al. (1986), a significant association was reported between pre-term delivery (before 37th week of pregnancy) and maternal blood lead levels at delivery for women living in a lead smelter town.

ACUTE/CHRONIC EFFECTS

The primary toxic effects associated with exposure to lead are alterations in the hematopoietic, nervous and cardiovascular systems. These effects have been extensively reviewed by EPA (1986b) and ATSDR (1988). The following is a summary of the major effects observed.

Lead exposure interrupts hemoglobin synthesis and may cause anemia at high exposure levels. Lead inhibits heme synthesis primarily by disturbing the activity of three major enzymes. Specifically, lead stimulates delta-aminolevulinic acid synthetase which mediates the production of delta-aminolevulinic acid (ALA), inhibits the activity of delta-aminolevulinic acid dehydrase, which catalyzes the conversion of ALA to porphyrobilinogen, and decreases the activity of ferrochelatase which catalyses the incorporation of iron into protoporphyrin to form heme. Decreased heme production results in decreased hemoglobin levels and can also have deleterious effects on other heme-containing proteins, such as cytochrome P_{450}, which detoxify certain chemicals in the body. No threshold has been identified for this effect on heme production. Impaired heme synthesis has been reported in adults at levels of less than 30 μg/dl lead in the blood (EPA, 1986b). Decreased delta-aminolevulinic acid dehydrase activity has been observed at blood lead levels as low as 3-5 μg/dl (ATSDR, 1988).

Two types of neurotoxic effects are associated with exposure to lead. Levels of lead in the blood (PbB) of over 80 μg/dl in children and over 120 μg/dl in adults can cause severe, irreversible brain damage, encephalopathy, and possibly death. Persons with these high blood levels may be asymptomatic or show only slight signs of intoxication, but rapid deterioration can occur. In children, permanent learning disabilities are seen at levels of 30 μg/dl, even if there are no overt signs of lead poisoning (EPA, 1984a; Goyer, 1988). Peripheral nerve function, as measured by nerve conduction velocity (NCV), was reported to be slowed at blood lead levels of <70 μg/dl and possibly as low as 30 μg/dl in lead workers (Seppäläinen et al., 1983).

Other adverse effects are associated with exposure to low levels of lead. Moderate increases in blood pressure have been reported in men with PbB levels of approximately 30 μg/dl; altered testicular function has been observed in men with PbB levels as low as 40-50 μg/dl; renal dysfunction has been associated with PbB levels as low as 40 μg/dl (EPA, 1986b).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

Ingestion of certain lead salts (lead acetate, lead subacetate) has been associated with an increased incidence of renal tumors in rats (EPA, 1985a), but a quantitative estimate of excess cancer risk has not been performed by the Carcinogen Assessment Group of EPA. EPA (1985b) noted that the available data provide an insufficient basis on which to regulate these compounds as human carcinogens. However, applying the criteria described in EPA's Guidelines for Carcinogenic Risk Assessment (EPA, 1986c), these lead salts have been classified by EPA (1985b) in Group B2—Probable Human Carcinogen. This category applies to agents for which there is inadequate evidence from human studies and sufficient evidence from animal studies.

Acceptable intakes for chronic or subchronic periods of exposure were not calculated for either inhalation or oral ingestion in the Health Effects Assessment Document (EPA, 1984) because the general population is already accruing unavoidable background exposures through food, water, and dust. EPA

considers that any significant increase above background exposure would represent a cause for concern.

The interim maximum contaminant level (MCL) for drinking water and the ambient water quality criterion (AWQC) are both 50 μg/liter (EPA, 1980; CFR, 1984). In deriving the ambient water quality criterion, EPA determined that consumption of water contaminated with 50 μg/liter would increase PbB levels to around 15 μg/dl in children. If the population average were 15 μg/dl, the Centers for Disease Control (CDC) calculated that 99% of all children would have a PbB level of less than 30 μg/dl. This was based on a CDC definition of "lead toxicity" in a child as a PbB level greater than or equal to 50 μg/dl. Based on this calculation, EPA determined that the present drinking water standard was acceptable as an upper limit. However, based primarily on the new information suggesting that neurological impairment may occur in children at PbB levels of less than 30 μg/dl, CDC has revised its criteria for lead toxicity to PbB levels greater than or equal to 25 μg/dl and erythrocyte protoporphyrin levels greater than or equal to 35 μg/dl (CDC, 1985).

A drinking water maximum contaminant level goal (MCLG) of 20 μg/liter has been proposed by the EPA Office of Drinking Water (ODW). The proposed MCLG is based upon the health effects of lead in infants and pregnant women which are considered sensitive subpopulations (EPA, 1985b). Blood levels above 15 μg/dl were identified as the level of concern, and fetuses and infants under 2 years of age are the most sensitive subpopulations. In order to protect the fetus, it was considered advisable to limit the blood lead level in women of child-bearing age to between 15 and 20 μg/dl, since studies indicate that the ratio of fetal/maternal blood lead values is close to 1:1 (Hubermont et al., 1978). An absorption constant of 0.16 was identified as the ratio of blood lead in infants to lead intake in the diet (Ryu et al., 1983). A reference dose (RfD) of 6×10^{-4} mg/kg/day can be derived from the proposed MCLG based on the ODW analysis. EPA's RfD Work Group recently discussed exposure to inorganic lead and lead compounds and considered it inappropriate to develop an RfD for inorganic lead (EPA, 1988) at this time.

The Clean Air Act National Ambient Air Quality Standard for lead is 1.5 $\mu g/m^3$. This standard is currently being evaluated for possible revision (EPA, 1985c).

At present, human health criteria for exposure to lead in soil have not been established in the United States. The United Kingdom Directorate of the Environment developed a tentative guideline for acceptable soil lead concentrations of 550 ppm in residential areas (Smith, 1981). Vernon Houk of the Centers for Disease Control has been quoted as indicating that soil lead concentrations of 300-400 ppm are acceptable based on studies of childhood lead poisoning (Mielke et al., 1984).

The American Conference of Governmental Industrial Hygienists (ACGIH, 1986) recommends a time-weighted average Threshold Limit Value (TLV) of 0.15 mg/m^3 lead in air.

SUMMARY OF LEAD CRITERIA

EPA carcinogen classification	Group B2
RfD	Not Available
MCL	50 μg/liter
Proposed MCL	5 μg/liter
NAAQS	1.5 $\mu g/m^3$

REFERENCES

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1988. Draft Toxicological Profile for Lead. U.S. Public Health Service; February 1988.

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio

AZAR, A., TROCHIMOWICZ, H.J., and MAXFIELD, M.E. 1973. Review of lead studies in animals carried out at Haskell Laboratory: Two year feeding study and response to hemorrhage study. In: Barth, D., Berlin, A., Engel, R., Recht, P., Smeets, J. eds. Environmental health aspects of lead: Proceedings, International Symposium, October 1972, Amsterdam, The Netherlands. Luxembourg: Commission of the European Communities; 199-210 (Cited in ATSDR, 1988)

BALO, J., BAJTAI, A., and SZENDA, B. 1965. Experimental adenomas of the kidney produced by chronic administration of lead phosphate. Magyar Onkil; 9: 144-151 (Cited in ATSDR, 1988)

BARLTROP, D. 1969. Transfer of lead to the human foetus. In: Barltrop, D., Burland, W.C., eds. Mineral metabolism in pediatrics. Philadelphia, PA, Davis Co. pp.135-151 (Cited in EPA, 1986a)

BARRY, P.S.I. 1975. A comparison of concentrations of lead in human tissues. Br. J. Ind. Med. 32: 119-139 (Cited in ATSDR, 1988)

BRUCE, W.R. and HEDDLE, J.A. 1979. The mutagenic activity of 61 agents as determined by the micronucleus, Salmonella and sperm abnormality assays. Can. J. Genet. Cytol.; 21: 319-334 (Cited in ATSDR, 1988)

CENTERS FOR DISEASE CONTROL (CDC). 1985. Preventing Lead Poisoning in Young Children. Public Health Service, Chronic Disease Division, Atlanta, Georgia. July 1985

CHAMBERLAIN, A., HEARD, C., LITTLE, M.J., et al. 1978. Investigations into lead from motor vehicles. Harwell, United Kingdom: United Kingdom Atomic Energy Authority. Report No. AERE-9198 (Cited in ATSDR, 1988)

CHOWDHURY, A.R., DEWAN, A., and GHANDI, D.N. 1984. Toxic effect of lead on the testes of rat. Biomed Biochim Acta; 43: 95-100. (Cited in ATSDR, 1988)

CODE OF FEDERAL REGULATIONS (CFR). 1984. 40 CFR 141, Environmental Protection Agency. National Interim Primary Drinking Water Regulations. U.S Government Printing Office, Washington, D.C.

DER, R., FAHIM, Z., HILDERBRAND, D., and FAHIM, M. 1974. Combined effect of lead and low protein diet on growth, sexual development, and metabolism in female rats. Res. Commun. Chem. Pathol. Pharmacol. 9:723-738 (As cited in EPA, 1986b)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Washington, D.C. December 1979. EPA 440/4-79-029

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Lead. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-057

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984b. Health Effects Asssessment for Lead. Environmental Criteria and Assessment Office. Cincinnati, Ohio. EPA 540/1-86-055

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Draft Health Advisory for Lead. Office of Drinking Water. Washington, D.C. September 30, 1985.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. National primary drinking water regulation; synthetic organic chemicals, inorganic chemicals and microorganisms; proposed rule. Fed. Reg. 50:46936-47025 (November 13, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985c. Draft air quality criteria document for lead: Notice of a corrigendum to the second external review draft. Fed. Reg. 50:14289-14294 (April 11, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985d. Water quality criteria; notice of final ambient water quality criteria documents. Fed. Reg. 50:30784-30796. (July 29, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986a. Air Quality Criteria for Lead. Volume III of IV. Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA 600/8-83/028cF

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986b. Air Quality Criteria for Lead. Volume IV of IV. Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA 600/8-83/028dF

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986c. Guidelines for carcinogenic risk assessment. Fed. Reg. 51: 33992-34003

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Integrated Risk Information System (IRIS). Chemical files on lead. On-line, Feb. 1989.

EVERSON, J. and PATTERSON, CC. 1980 "Ultra-clean" isotope dilution/mass spectrometric analyses for lead in human blood plasma indicate that most reported values are artificially high. Clin. Chem: 26:1603-1607

FAHIM, M.S., FAHIM, Z., and HALL, D.G. 1976. Effects of subtoxic lead levels on pregnant women in the State of Missouri. Res. Commun. Chem. Pathol. Pharmacol.; 13:309-331

GUY, R.D. and CHAKRABARTI, C.L. 1976. Studies of metal-organic interactions in model systems pertaining to natural waters. Can. J. Chem 54:2600-2611

HAMMOND, P.B. 1982. Metabolism of lead. In: Chisolm, J.J., O'Hara, D.M., eds. Lead absorption in children: management, clinical and environmental aspects. Baltimore, MD: Urban and Schwarzenberg: 11-20 (Cited in EPA, 1986a)

HILDERBRAND, D.C., DER, R., GRIFFIN, W.T., and FAHIM, M.S. 1973. Effect of lead acetate on reproduction. Am. J. Obstet. Gynecol.; 115:1058-1065 (Cited in EPA, 1986b)

HOFMANN, W. 1982. Dose calculations for the respiratory tract from inhaled natural radioactive nuclides as a function of age-II. Basal cell dose distributions and associated lung cancer risk. Health Phys. 43:31-44

HORIGUCHI, S. and UTSUNOMIYA, T. 1973. An estimate of the body burden of lead in the healthy Japanese population: an attempt to assume absorption and excretion of lead in the health Japanese population, part 2. Osaka City Med. J. 19:1-5 (Cited in EPA, 1986a)

HORIUCHI, K., HORIGUCHI, S., and SUEKANE, M. 1959. Studies on industrial lead poisoning. 1: Absorption, transportation, deposition and excretion of lead. 6: The lead contents in organ-tissues of the normal Japanese. Osaka City Med. J.; 5:41-70. (Cited in EPA, 1986a)

HUANG, C.P., ELLIOTT, H.A., and ASHMEAN, R.M. 1977. Interfacial reactions and the fate of heavy metals in soil water systems. J. Water Pollut. Control 49:745-756

HUBERMONT, G., BUCHET, J.P., ROELS, H., and LAUWERYS, R. 1978. Placental transfer of lead, mercury and cadmium in women living in a rural area: importance of drinking water in lead exposure. Int. Occup. Environ. Health. 41:117-124. (Cited in EPA, 1985a)

INDRAPRASIT, S., ALEXANDER, G.V., and GONICK, H.C. 1974. Tissue composition of major and trace elements in uremia and hypertension. J. Chronic Dis.; 27:135-161 (Cited in EPA, 1986a)

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1982. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Supplement 4: Chemicals, Industrial Processes and Industries Associated with Cancer in Humans. World Health Organization, Lyon, France. pp. 149-150

KORTE, N.E., SKOPP, J., FULLER, W.H., NIEVLA, E.E., and ALESII, B.A. 1976. Trace element movement in soils: Influence of soil physical and chemical properties. Soil Sci. 122:350-525

KRASOVSKII, G.N., VASUKOVICH, L.Y., and CHARIEV, O.G. 1979. Experimental study of biological effects of lead and aluminum following oral administration. Environ. Health Perspect.; 30:47-51 (Cited in EPA, 1986b)

LEONARD A., GERBER, G.B., JACQUET, P., and LAUWERYS, R.R. 1984. Mutagenicity, Carcinogenicity and Teratogenicity of Industrially Used Metals. In: Mutagenicity, Carcinogenicity and Teratogenicity of Industrial Pollutants. Kirsch-Volders, M., (ed.), Plenum Press, New York and London. pp. 59-126

MAISIN, J.R., JADE, J.M., and LAMBIER-COLLIER, M. 1975. Progress report on morphological studies of the toxic effects of lead on the reproductive organs and embryos. Brussels, Belgium: Economic Community of Europe. Contract No. 080-74-7; Env. B. (As cited in EPA, 1986b)

McMICHAEL, A.J., VIMPANI, G.V., ROBERTSON, E.F., BAGHURST, P.A., and CLARK, P.D. 1986. The Port Pirie cohort study: maternal blood lead and pregnancy outcome. J. Epidem. Commun. Health; 40:18-25 (As cited in ATSDR, 1988).

MIELKE, H.W., BLAKE, B., BURROUGHS, S., and HASSINGER, N. 1984. Urban lead levels in Minneapolis: The case of the Hmong children. Environ. Rev. 34:64-76. (As cited in EPA, 1985b)

NATIONAL ACADEMY OF SCIENCES. 1980. Lead in the Human Environment; Report prepared by Committee on Lead in the Human Environment. Washington, D.C.

NORDSTROM, S., BECKMAN, L., and NORDENSON, I. 1979. Occupational and environmental risks in and around a smelter in northern Sweden: V. spontaneous abortion among female employees and decreased birth weight in their offspring. Hereditas 90:293-296 (Cited in EPA, 1986b)

NORDSTROM, S., BECKMAN, L., and NORDENSON, I. 1978. Occupational and environmental risks in and around a smelter in northern Sweden: I. Variations in birth weight. Hereditas 88:43-46 (Cited in EPA, 1986b)

RYU, J.E., ZIEGLER, E.E., NELSON, F.E., and FOMON, S.J. 1983. Dietary intake of lead and blood lead concentration in early infancy. Am. J. Dis. Child. 137:886-891. (As cited in EPA, 1985b)

SCHROEDER, H.A. and MITCHENER, M. 1971. Toxic effects of trace elements on the reproduction of mice and rats. Arch. Environ. Health 23:102-106 (As cited in EPA, 1986b)

SCHROEDER, H.A. and TIPTON, I.H. 1968. The human body burden of lead. Arch. Environ. Health 7:965-978

SEPPÄLÄINEN, A.M., HERNBERG, S., VESANTO, R., and KOCK, B. 1983. Early neurotoxic effects of occupational lead exposure: a prospective study. Neurotoxicology; 4:181-192 (As cited in ATSDR, 1988).

SMITH, M.A. 1981. Tentative Guidelines for Acceptable Concentrations of Contaminants in Soils. Department of the Environment, United Kingdom

STOWE, H.D. and GOYER, R.A. 1971. The reproductive ability and progeny of F_1 lead-toxic rats. Fertil. Steril. 22:755-760 (As cited in EPA, 1986b)

STOWE, H.D., GOYER, R.A., KRIGMAN, M.M., WILSON, M., and CATES, M. 1973. Experimental oral lead toxicity in young dogs: clinical and morphological effects. Arch. Pathol. 95:106-116 (As cited in EPA, 1986b)

VAN ESCH, G.J., VAN GENDEREN, H., and VINK, H.H. 1962. The induction of renal tumors by feeding of basic lead acetate to rats. Br. J. Cancer; 16:289-297. (Cited in EPA, 1986b)

VAN ESCH, G.J. and KROES, R. 1969. The induction of renal tumors by feeding basic lead acetate to mice and hamsters. Br. J. Cancer; 23:765-771

VARMA, M.M., JOSHI, S.R., and ADEYEMI, A.O. 1974. Mutagenicity and infertility following administration of lead sub-acetate to Swiss male mice. Experientia 30:486-487 (As cited in EPA, 1986b)

WONG, P.T.S., CHAN, Y.K., and LUXON, P.L. 1975. Methylation of lead in the environment. Nature. 253:263-264

ZOLLINGER, H.U. 1953. Kidney adenomas and carcinomas in rats caused by chronic lead poisoning and their relationship to corresponding human neoplasms. Virchows Arch. Pathol. Anat. Physiol.; 323:694-710. (Cited in EPA, 1986a)

A.6 POLYCHLORINATED BIPHENYLS (PCBs)

INTRODUCTION

PCBs are complex mixtures of chlorinated biphenyls. The commercial PCB mixtures that were manufactured in the United States were given the trade name of "Aroclor." Aroclors are distinguished by a four-digit number (for example, Aroclor 1260). The last two digits in the Aroclor 1200 series represent the average percentage by weight of chlorine in the product.

Concentrations of PCBs reported in ambient air range from 0.02-0.5 ng/m^3 in remote locations to 0.1-2.0 ng/m^3 in rural locations and 0.5-3.0 ng/m^3 in urban areas. The low solubility of PCBs generally limits their concentrations in drinking water supplies (EPA, 1980). Mean PCB concentrations range from approximately 1-20 ng/liter (Baker et al., 1985; Rodgers and Swain, 1983).

TOXICOKINETICS

ABSORPTION

PCBs are readily absorbed through the gastrointestinal (G.I.) tract and somewhat less readily through the skin. G.I. absorption in rats is greater than 90% for a wide range of isomers (Albro and Fishbein, 1972). Dermal absorption of Aroclor 1242 in a benzene/hexane solution has been reported to be 15-34% in monkeys (Wester et al., 1983). PCBs are presumably readily absorbed from the lungs, but few data are available that experimentally define the extent (EPA, 1985a).

METABOLISM AND EXCRETION

PCBs are distributed through the bloodstream initially to the liver, but within a few days liver concentrations are greatly reduced as redistribution and metabolism occur. Lesser chlorinated chlorobiphenyl isomers are

metabolized, but chlorobiphenyl isomers with higher percentages of chlorine tend to redistribute to other organs, adipose tissue, and skin. Sequestering of PCBs in adipose tissue isolates them from metabolically active liver enzymes and greatly retards their clearance from the body.

The major metabolic products of PCBs are phenolic derivatives or dihydrodiols. Conjugated PCB metabolites are excreted in urine and bile. Excretion in urine is more prominent for lesser chlorinated chlorobiphenyl isomers, while bile becomes significant for more highly chlorinated compounds (Safe, 1980). Excretion of PCBs also has been reported in milk (Deichmann, 1981).

ENZYME INDUCTION

Like other organochlorine compounds, PCBs can induce microsomal enzymes. Microsomal mixed-function oxidase enzymes metabolize a wide variety of lipophilic compounds. In general, the intensity of induction increases with increasing chlorination of the PCBs and is dose-related. The enhanced activity of hepatic enzymes induced by PCBs can persist for a considerable period of time after dosing (NRCC, 1978).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

The toxic effects of PCBs in humans and other mammals are summarized in the following subsections.

CARCINOGENIC POTENTIAL

A number of studies have suggested that PCB mixtures are capable of increasing the frequency of tumors in animals exposed for long periods of time. Exposure of female rats to 0 or 100 ppm Aroclor 1260 in the diet for 20-21 months resulted in a high frequency of liver tumors in treated animals (Kimbrough et al., 1975). The National Cancer Institute (NCI, 1978) reported significant dose-related increases of liver tumors and hepatocellular

hyperplastic nodules in rats fed diets containing 0, 25, 50, or 100 ppm Aroclor 1254 for 104-105 weeks. Schaeffer et al. (1984) reported results of a comparative study of the carcinogenicity and chronic toxicity of Clophen A60 (equivalent to Aroclor 1260) and Clophen A30 (equivalent to Aroclor 1242) in male rats exposed to concentrations of 100 ppm in the diets for up to 832 days. A strong carcinogenic effect of Clophen A60 (hepatocellular carcinomas and/or neoplastic nodules) and a weaker carcinogenic effect of Clophen A30 (hepatocellular neoplastic nodules) were reported. Norback and Weltman (1985) exposed male and female rats to a diet containing Aroclor 1260 at a concentration of 100 ppm for 16 months, 50 ppm for an additional 8 months, and control diet for 5 months. Liver tumors were produced in females, with a much weaker response in males.

Other studies have suggested that PCB mixtures can also promote or inhibit the action of other carcinogens in rats and mice. Preston et al. (1981) reported that exposure to Aroclor 1254 at a concentration of 100 ppm in the diet for 18 weeks markedly augmented the development of liver tumors initiated by diethylnitrosamine in rats. In two-stage bioassay systems using diethylnitrosamine as an initiator, Clophen A50 (equivalent to Aroclor 1254) and Aroclor 1254 were shown to be promoters for rat liver tumors by Oesterle and Deml (1984) and Pereira et al. (1982), respectively. The Oesterle and Deml (1984) study is the only dose-response study of cancer promotion by PCBs.

GENOTOXIC POTENTIAL

PCB mixtures and several isomers have been studied for genotoxic effects, and the results have been generally negative. ATSDR (1987) reviewed several studies in which PCBs did not induce mutations in bacteria or mammalian cells treated in vitro and they did not induce dominant lethal mutations in mice exposed in vivo. Weakly positive results for chromosome aberrations were reported in doves exposed to Aroclor 1254, but negative results were produced in other in vivo tests in mice, rats, chickens and Drosophila and in an in vitro test with human lymphocytes.

Studies of the metabolic activation of PCBs and their ability to induce microsomal enzymes have demonstrated that PCB isomers and their metabolites bind to macromolecules, including DNA, with varying degrees of affinity (EPA, 1985a). This suggests that PCBs are capable of damaging DNA, but it is unclear whether the damage is repairable or likely to produce permanent alterations in the genome. Induction of enzymes responsible for the metabolic activation of other known mutagens and carcinogens could result in enhanced effects of these other agents.

REPRODUCTIVE EFFECTS

Reproductive outcomes of pregnant women consuming fish heavily contaminated with PCBs from Lake Michigan have been compared to births from women who reported no such exposure (Fein et al., 1984a,b; Jacobson et al., 1983; Jacobson et al., 1984). Reduced birth weights, slow weight gain, reduced gestational ages, and behavioral deficits in infants were reported in this study. The study did not, however, rigorously establish that the causative factor was exposure to PCBs rather than other contaminants present in Lake Michigan fish.

Reproductive effects of PCBs have been demonstrated in animals, but there appear to be major differences in the susceptibility of different species. Mink and monkeys appear to be relatively sensitive; mice, rats, and rabbits appear relatively insensitive.

Dietary administration of Aroclor 1254 at 2 ppm or Aroclor 1243 at 5 ppm for 9 months prior to whelping has been reported to inhibit reproductive success in mink. Aroclor 1016 at concentrations as high as 20 ppm did not lower the number of young born per female. The PCBs were embryotoxic to developing embryos, but spermatogenesis, oogenesis, and implantation were unaffected. Transfer of PCBs to the surviving pups through mother's milk was a significant contributing source of exposure for the offspring (Ringer, 1983).

Monkeys also appear to be sensitive to the reproductive effects of PCBs. Altered menstrual cycles, reduced breeding success, and lowered birth rates were reported in monkeys exposed to dietary levels of 2.5 and 5.0 ppm Aroclor 1248 for 6 to 7 months prior to breeding (Barsotti et al., 1976). When female monkeys were exposed to 0.5 or 1 ppm Aroclor 1248 (three times per week) for 7 months prior to breeding (Allen et al., 1979; Bowman et al., 1981), no toxic signs were observed in the mothers. The infants, however, had reduced birth weights, developed hyperpigmentation during nursing, and were marginally hyperactive at one year of age. Reduced birth weights were also reported in the offspring of female monkeys fed diets containing 1.0 ppm Aroclor 1016 for about 7 months prior to mating and during pregnancy. No fetotoxic effects were observed in the offspring of monkeys fed diets containing 0.25 ppm on the same schedule (Barsotti and Van Miller, 1984). For comparison, Linder et al. (1974) performed a two-generation study in rats and reported dose-related effects (reduced numbers of litters, litter-sizes and pup survivals, and pathological lesions in offspring) for Aroclor 1254 at dietary concentrations of 20 ppm. Aroclor 1260 was less toxic than Aroclor 1254 in the same experiment.

ACUTE/CHRONIC EFFECTS

In considering the health effects of PCB exposure in humans, it is important to note that PCBs are often contaminated with highly toxic impurities, particularly polychlorinated dibenzofurans (PCDFs). Effects of these two chemical classes have not been identified in most human studies and the following discussion refers to commercial mixtures. The reader should recognize that at least some of the reported effects may be due to the PCDF impurities.

Humans

Dermatitis and chloracne (a disfiguring and long-term skin disease) have been the most prominent and consistent finding in studies of occupational exposure to PCBs. Interpretation of these data is complicated by the

difficulty of diagnosing chloracne and the uncertainties of blood PCB determinations. Reports of both chloracne and other PCB-related skin effects have generally been associated with exposures to more highly chlorinated PCB mixtures containing 42% chlorine or more (Chase et al., 1982; Emmett et al., 1983; Maroni et al., 1981a,b; Fishbein et al., 1979). Smith et al. (1982) reported no PCB-related skin effects in workers exposed to high levels of Aroclor 1016, which is less chlorinated.

Several studies examining liver function in exposed humans have reported increased blood levels of liver enzymes. Maroni et al. (1981b), Chase et al. (1982), Smith et al. (1982), and Emmett et al. (1983) reported statistically significant associations between blood PCB levels and elevated levels of liver enzymes. A "no-effect" level was not identified for these effects, as associations were found in all individuals with detectable blood levels of PCBs.

Animals

Based on the published literature, reproductive, hepatic, and immunotoxic effects appear to be the most sensitive endpoints of PCB toxicity in nonrodent species, and the liver appears to be the most sensitive target organ for toxicity in rodents.

PCBs are not highly toxic when given as a single oral dose to mammals (Kimbrough et al., 1978), and would be classified as only slightly toxic based on their acute oral toxicity (Hodge and Sterner, 1949). The more significant toxic effects of PCBs are observed after repeated exposure over a period of time.

The most consistent pathological changes associated with PCB exposure in mammals other than monkeys and mink are in the liver (EPA, 1985a). Fatty deposits and increased liver sizes are commonly reported. Individual hepatocytes may appear foamy and vacuolated. Cellular necrosis can occur at high doses, and the location of lesions is often centrilobular (Sleight, 1983). Comparative studies have shown that Aroclor 1254 is usually the most

hepatotoxic mixture. In rats, Aroclor 1260 was significantly less toxic to the liver than Aroclor 1254 (Kimbrough et al., 1972). Aroclors 1248, 1242, and 1016 are progressively less toxic than Aroclor 1254 (Koller, 1977; Burse et al., 1974).

A skin syndrome similar to the effects seen in humans has been reported in monkeys. Barsotti et al. (1976) and Allen et al. (1979) fed diets containing 2.5 and 5 ppm Aroclor 1248 to rhesus monkeys for 7 months. Weight loss, followed by hair loss, acne on the face and neck, eyelid edema and erythema, and keratinization of hair follicles were reported. McNulty et al. (1980) reported similar effects in rhesus monkeys exposed to Aroclor 1242 and individual chlorobiphenyl isomers. These authors indicated that swelling of the eyelids, resulting from dilation of the Meibomian glands is the most sensitive indicator of chlorinated aromatic hydrocarbon poisoning of monkeys.

Immunotoxic and immunosuppressive effects have been reported in most experiments in which these endpoints have been investigated, and are among the more sensitive indicators of PCB exposure (EPA, 1985a). Monkeys seem to be the most sensitive species; guinea pigs are relatively sensitive; and rabbits, rats and mice appear relatively insensitive. Rhesus monkeys chronically exposed to Aroclor 1248 exhibited atrophy or hypocellularity of the thymus, spleen, lymph nodes, and bone marrow; these effects were particularly marked in infants after chronic maternal exposure to diets containing 2.5 ppm Aroclor 1248 (Allen and Barsotti, 1976; Allen et al., 1980). Reductions in cell-mediated immunity, humoral immunity, or both were reported in monkeys chronically exposed to diets containing 1.5 or 5 ppm Aroclor 1248 (Thomas and Hinsdill, 1978), and in monkeys dosed with 100 or 400 μg/kg/day Aroclor 1254 (Truelove et al., 1982). Increased susceptibilities to infectious diseases in PCB-exposed monkeys have been reported by Allen et al. (1979), McNulty et al. (1980), and Barsotti et al. (1976).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

EPA (1984) classified PCBs in Group B2—Probable Human Carcinogen based on sufficient evidence in animal bioassays and inadequate evidence from studies in humans. EPA (1987a) recommended that all commercial PCB mixtures be considered to have a similar carcinogenic potential.

EPA (1987a) calculated an oral cancer potency factor for PCBs based on a study of rats exposed to a dose of 5 mg/kg/day Aroclor 1260 (Norback and Weltman, 1985). The data on liver tumor incidence in female rats (1/49 in controls and 45/47 in treated animals) was used to calculate the 95% upper confidence limit on risk. The risks determined using this approach are unlikely to underestimate the actual risks posed by exposure to low levels of PCBs in the environment and may overestimate risk. The carcinogenic potency factor for lifetime exposure to PCBs is 7.7 $(mg/kg/day)^{-1}$. This potency estimate has been verified by EPA (1987b) and it is assumed to apply to all PCB mixtures (EPA, 1987a). It has not yet been entered into EPA's Integrated Risk Information System (IRIS) database.

The American Conference of Governmental Industrial hygienists (ACGIH, 1986) recommends a time-weighted average Threshold Limit Value (TLV) of 1 mg/m^3 for Aroclor 1242 as protection against systemic intoxication. The ACGIH also recommends a time-weighted average TLV of 0.5 mg/m^3 for Aroclor 1254.

SUMMARY OF PCB CRITERIA

EPA Carcinogen Classification	Group B2
Oral Carcinogenic Potency Factor	7.7 mg/kg/day
RfD	Not Available
MCL	None Available
Proposed MCLG	0 mg/l

REFERENCES

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987. Draft Toxicological Profile for Selected PCBs. U.S. Public Health Service; November 1987

ALBRO, P.W. and L. FISHBEIN. 1972. Intestinal absorption of polychlorinated biphenyls in rats. Bull. Environ. Contam. Toxicol. 8:26

ALLEN, J.R. and BARSOTTI, D.A. 1976. The effects of transplacental and mammary movement of PCBs on infant rhesus monkeys. Toxicology 6:331-340

ALLEN, J.R., HARGRAVES, W.A., HSIA, M.T., et. al. 1979. Comparative toxicology of chlorinated compounds on mammalian species. Pharmacol. Ther. 7:513-547

ALLEN, J.R., BARSOTTI, D.A., and CARSTENS, L.A. 1980. Residual effects of polychlorinated biphenyls on adult nonhuman primates and their offspring. J. Toxicol. Environ. Health 6:55-66

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio

BAKER, J.E., EISENREICH, S.J., JOHNSON, T.C., and HALFMAN, B.M. 1985. Chlorinated hydrocarbon cycling in the benthic nepreloid layer of Lake Superior. Environ. Sci. Technol.; 19: 854-61 (As cited in ATSDR, 1987)

BARSOTTI, D.A., MARLAR, F.J., and ALLEN, J.R. 1976. Reproductive dysfunction in rhesus monkeys exposed to low levels of polychlorinated biphenyls (Aroclor 1248). Food Cosmet. Toxicol. 14:99

BARSOTTI, D.A. and VAN MILLER, J.P. 1984. Accumulation of a commercial polychlorinated biphenyl mixture (Aroclor 1016) in adult rhesus monkeys and their nursing infants. Toxicology; 30(1): 31-44 (As cited in ATSDR, 1987)

BOWMAN, R.E. and HEIRONIMUS, M.P. 1981. Hypoactivity in adolescent monkeys perinatally exposed to PCBs and hyperactive as juveniles. Neurobeh. Toxicol. Teratol. 3:15-18

BURSE, V.W., KIMBROUGH, R.D., VILLANUEVA, E.C., JENNINGS, R.W., LINDER, R.E., and SOVOCOOL, G.W. 1974. Polychlorinated biphenyls-Storage, distribution, excretion and recovery: Liver morphology after prolonged dietary ingestion. Arch. Environ. Health 29:301-307

CHASE, K.H., WONG, O., THOMAS, D., BERNEY, B.W., and SIMON, R.K. 1982. Clinical and metabolic abnormalities associated with occupational exposure to polychlorinated biphenyls. J. Occup. Med. 24:109-114

DEICHMANN, W.B. 1981. Halogenated cyclic hydrocarbons. In Clayton, G.D., and Clayton, F.E., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York

EISENREICH, S.J., LOONEY, B.B., and THORTON, J.D. 1981. Airborne organic contaminants in the Great Lakes ecosystem. Environ. Sci. Technol., 15:30-38

EMMETT, E.A., AGNEW, J., BLEEKER, M.L., FERRARA, J.M., LEVIN, B.K., JEFFREYS, J., and MARONI, M. 1983. Health Effects of PCB Exposure of GSA Switchgear Employees. NIOSH Health Hazard Evaluation Program Medical Report, Technical Assistance Request No. 80-7. The Johns Hopkins University, Baltimore, Maryland

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Washington, D.C. December 1979. EPA 440/4-79-029

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Polychlorinated Biphenyls. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA/440/5-80-068. NTIS PB 81-117798

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Polychlorinated Biphenyls. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-004

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Health Effects Criteria Document on Polychlorinated Biphenyls. Office of Drinking Water. Washington, Final Draft

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. National primary drinking water regulations; synthetic organic chemicals, inorganic chemicals and microorganisms. Fed. Reg. 50:47001-47002. (November 13, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987a. Drinking Water Criteria Document for Polychlorinated Biphenyls (PCBs). ECAO-CIN-414. Final. (As cited in ATSDR, 1987)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987b. IRIS (Integrated Risk Information System), CRAVE (Chronic Risk Assessment Validation Endeavor) for polychlorinated biphenyls. (Verification date: 4/22/87). Online: input pending. Cincinnati, OH: OHEA, ECAO. (As cited in ATSDR, 1987)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987c. Graphical exposure modeling system (GEMS). Personal computer version April, 1987. Research Triangle Park, NC: EPA (As cited in ATSDR, 1987)

FEIN, G.G., JACOBSON, J.L., JACOBSON, S.W., SCHWARTZ, P.M., and DOWLER, J.K. 1984a. Prenatal exposure to polychlorinated biphenyls: Effects on birth size and gestational age. J. Pediatr. 105:315-320

FEIN, G.G., JACOBSON, J.L., JACOBSON, S.W., and SCHWARTZ, P.M. 1984b. Intrauterine Exposure of Humans to PCBs: Newborn Effects NTIS BP84-188887, EPA 600/3-84-060

FISCHBEIN, A., WOLFF, M.S., LILIS, R., THORNTON, J., and SELIKOFF, I.J. 1979. Clinical finding among PCB-exposed capacitor manufacturing workers. Ann. NY Acad. Sci. 320:703

GRIFFIN, R.A. and CHOU, S.F.J. 1981. Movement of PCBs and other persistent compounds through soil. Water Sci. Technol., 13:1153-1163

HAWKER, D.W. and CONNELL, D.W. 1988. Octanol-water partition coefficients of polychlorinated biphenyl congeners, Envir. Sci. and Technol. 22(4):382-387

HODGE, H.C. and STERNER, J.H. 1949. Tabulation of toxicity classes. Am. Ind. Hyg. Assoc. Q. 10:93

JACOBSON, S.W., JACOBSON, J.L., SCHWARTZ, P.M., and FEIN, G.G. 1983. Intrauterine exposure of human newborns to PCBs: Measures of exposure. In D'Itri, F.M., and Kamrin, M.A., eds., PCBs: Human and Environmental Hazards. Butterworth Publishers, Boston. pp. 311-343

JACOBSON, J.L., FEIN, G.G., JACOBSON, S.W., SCHWARTZ, P.M., and DOWLER, J.K. 1984. The transfer of polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PBBs) across the human placenta and into maternal milk. Am. J. Public Health 74:378-379

KIMBROUGH, R.D., LINDER, R.E., and GAINES, T.B. 1972. Morphological changes in livers of rats fed polychlorinated biphenyls. Arch. Environ. Health 35:354

KIMBROUGH, R.D., SQUIRE, R.A., LINDER, R.E., STRANDBERG, J.D., MONTALI, R.J., and BURSE, V.W. 1975. Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. J. Natl. Cancer Inst. 55:1453

KIMBROUGH, R. et al. 1978. Animal toxicity. Environ. Health Perspect. 24:173

KOLLER, L.D. 1977. Enhanced polychlorinated biphenyl lesions in Moloney leukemia virus-infected mice. Clin. Toxicol. 11:107-116

LEIFER, A., BRINK, R.H., THOM, G.C., and PARTYMILLER, K.G. 1983. Environmental transport and transformation of polychlorinated biphenyls. Washington, D.C.: Office of Pest. Tox. Sub., EPA-560/5-83-025

LINDER, R.E., GAINES, T.B., and KIMBROUGH, R.D. 1974. The effect of PCB on rat reproduction. Food Cosmet. Toxicol. 12:63

MACKAY, D. and LEINONEN, P.J. 1975. Rate of evaporation of low-solubility contaminants from water bodies to the atmosphere. Environ. Sci. Technol. 9(13):1178-1180

MARONI, M., COLOMBI, A., CANTONI, S., FERIOLI, E., and FOA, V. 1981a. Occupational exposure to polychlorinated biphenyls in electrical workers. I. Environmental and blood polychlorinated biphenyls concentrations. Br. J. Ind. Med. 38:55-60

MARONI, M., COLUMBI, A., ARBOSTI, G., CANTONI, S., and FOA, V. 1981b. Occupational exposure to polychlorinated biphenyls in electrical workers. II. Health effects. Br. J. Ind. Med. 38:55-60

MATTHEWS, H.B. 1983. Metabolism of PCBs in Mammals: Routes of Entry, Storage and Excretion. In D'Itri, F.M., and Kamrin, M.A., eds. PCBs: Human and Environmental Hazards. Butterworth, Boston.

McNULTY, W.P., BECKER, G.M., and CORY, H.T. 1980. Chronic toxicity of 3,4,3',4- and 2,5,2',5'-tetrachlorobiphenyls in rhesus macaques. Toxicol. Appl. Pharmacol. 56:182-190

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of Aroclor 1254 for Possible Carcinogenicity. CAS. No. 27323-18-8. NCI Carcinogenesis Technical Report Series No. 38. DHEW Publication No. (NIH)78-838

NATIONAL RESEARCH COUNCIL OF CANADA (NRCC). 1978. Polychlorinated biphenyls: Biological criteria for an assessment of their effects on environmental quality. NRCC. No. 16077. Ottawa, Ontario.

NORBACK, D.H. and WELTMAN, R.H. 1985. Polychlorinated biphenyl induction of hepatocellular carcinoma in the Sprague-Dawley rat. Environ. Health Perspect.; 60: 97-105 (As cited in ATSDR, 1987)

OESTERLE, D. and DEML, E. 1984. Dose-dependent promoting effect of polychlorinated biphenyls on enzyme-altered islands in livers of adult and weanling rats. Carcinogenesis 5:351-355

PEREIRA, M.A., HERREN, S.L., BRITT, A.L., and KHOURY, M.M. 1982. Promotion of polychlorinated biphenyls of enzyme altered foci in rat liver. Cancer Letters 15:185-190

PRESTON, B.D., VAN MILLER, J.P., MOORE, R.W., and ALLEN, J.R. 1981. Promoting effects of polychlorinated biphenyls (Aroclor 1254) and polychlorinated dibenzofuran-free Aroclor 1254 on diethylnitrosamine-induced tumorigenesis in the rat. J. Natl. Canc. Inst. 66:509-515

RINGER, R.K. 1983. Toxicology of PCBs in Mink and Ferrets. In D'Itri, F.M., and Kamrin, M.A., eds. PCBs: Human and Environmental Hazards. Butterworth, Boston.

RODGERS, P.W. and SWAIN, W.R. 1983. Analysis of polychlorinated biphenyl (PCB) loading trends in Lake Michigan. J. Great Lakes Res.; 9: 548-58

SAFE, S. 1980. Halogenated biphenyls, terphenyls, naphthalenes, dibenzodioxins and related products. Metabolism, uptake, storage, and bioaccumulation. Toxicol. Environ. Health 4:81-107

SCHAEFFER, E., GREIM, H., and GOESSNER, W. 1984. Pathology of chronic polychlorinated biphenyl (PCB) feeding in rats. Toxicol. Appl. Pharmacol. 75:278-288

SLEIGHT, S.D. 1983. Pathologic effects of PCBs in mammals. In D'Itri, F.M., and Kamrin, M.A., eds. PCBs: Human and Environmental Hazards. Butterworth, Boston.

SMITH, A.B. et al. 1982. Metabolic and health consequences of occupational exposure to polychlorinted biphenyls. Br. J. Ind. Med. 39:361-369

THOMAS, P.T. and HINSDILL, R.D. 1978. Effect of polychlorinated biphenyls on the immune responses of rhesus monkeys and mice. Toxicol. Appl. Pharmacol. 44:41-51

TRUELOVE, J., GRANT, D., MES, J., TRYPHONAS, H., and ZAWIDZKA, Z. 1982. Polychlorinated biphenyl toxicity in the pregnant cynomolgus monkey: A pilot study. Arch. Environ. Contam. Toxicol. 11:583-588

WEAST, R.E., ed. 1983. Handbook of Chemistry and Physics. 63rd ed. CRC Press, Cleveland, Ohio

WESTER, R.C., BUCKS, A.W., MAIBACK, H.I., and ANDERSON, J. 1983. Polychlorinated Biphenyls (PCBs): Dermal Absorption, Systemic Elimination, and Dermal Wash Efficiency. J. Toxicol Environ. Hlth. 12:511

A.7 POLYCYCLIC AROMATIC HYDROCARBONS

INTRODUCTION

Polycyclic aromatic hydrocarbons (PAHs) are a diverse class of compounds consisting of two or more fused aromatic rings. They are formed during the incomplete combustion of materials containing carbon and hydrogen and are ubiquitous in the modern environment. PAHs are commonly found as constituents of coal tar, soot, vehicular exhausts, cigarette smoke, certain petroleum products, road tar, mineral oils, creosote, and many cooked foods. The occurrence of individual PAHs in these materials and typical environmental concentrations are reviewed in IARC (1983).

A number of reviews have been prepared on the toxicology of the PAHs. The Environmental Protection Agency (EPA) prepared an Ambient Water Quality Criteria Document on the general class of polycyclic aromatic hydrocarbons (EPA, 1980a) and also prepared Criteria Documents on several specific PAHs, including acenaphthene, fluoranthene, and naphthalene (EPA, 1980b-d). More recently, EPA (1984a-f) prepared Health Effects Assessments for PAHs as a class, for coal tars, and for the individual compounds benzo(a)pyrene (B(a)P), naphthalene, phenanthrene, and pyrene. In addition to the EPA documents, Santodonato et al. (1981) prepared a review and risk assessment of polycyclic aromatic hydrocarbons, the International Agency for Research on Cancer (IARC) reviewed the toxicity and carcinogenicity of a large number of individual PAHs and PAH-containing mixtures (IARC, 1973, 1983, 1984, 1985), and the Agency for Toxic Substances and Disease Registry (ATSDR) prepared toxicological profiles for benz(a)anthracene, benzo(a)pyrene, benzo(b)fluorene, and dibenzo(a,h)anthracene (ATSDR, 1987a-d).

For practical purposes, the PAHs are often separated into two categories, the "carcinogenic" and the "noncarcinogenic" PAHs. This is a somewhat misleading categorization as many of the "noncarcinogenic" PAHs have been shown to have some, albeit weak, carcinogenic activity, or to act as promoters or co-carcinogens. A more accurate designation would be to

differentiate between potent carcinogens, weak carcinogens, and noncarcinogens.

A number of factors have been shown to influence the relative carcinogenic potencies of the PAHs. These include planarity of the molecule, cellular absorption, binding affinity, the presence or absence of a particular molecular structure, and the electron configuration of the molecule (Dipple et al., 1984; Frierson et al., 1986). In addition, genetic differences in the exposed animals, particularly in their ability to produce the enzyme aryl hydrocarbon hydroxylase (AHH), have been shown to influence carcinogenic potency. Finally, PAHs are not ultimate carcinogens but must be metabolized before they become biologically activated. A complete description of the complex metabolism of this class of compounds is beyond the scope of this report, but a detailed review of the factors influencing the carcinogenicity of the PAHs and metabolism of these compounds can be found in Dipple et al. (1984), Santodonato et al. (1981), and Frierson et al. (1986)

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

A number of PAHs have been shown to be potent animal carcinogens, producing tumors both at the site of application and systemically, in several different animal species, when administered by any of a number of routes. For example, Rigdon and Neal (1969) reported gastric tumors, pulmonary adenomas and leukemias in mice fed benzo(a)pyrene, and intratracheal instillation of a number of PAHs has been shown to cause lung tumors in both mice and hamsters (Santodonato et al., 1981). In addition, IARC (1984, 1985) noted that occupational exposure to coal soot, coal tar and pitch, coal tar fumes, and some impure mineral oils causes cancer in humans at several sites, including the skin, and concluded that there is sufficient evidence that soot, tars, and some mineral oils are carcinogenic in humans. Fractionation procedures have demonstrated that the PAHs are the carcinogenic agents in coal tar. Nonneoplastic lesions are seen in animals exposed to the more potent carcinogenic PAHs only after exposure to levels well above those required to elicit a carcinogenic response.

Acute effects from direct contact with PAHs and related materials are limited primarily to phototoxicity. Phototoxicity is caused by exposure to a toxic substance followed by exposure to sunlight. The primary effect is dermatitis -- skin reddening, itching, and burning. NIOSH (1977) reviewed the phototoxic effects of exposure to coal tar and found that phototoxicity can result from a single 90-minute exposure to a 1% solution of coal tar. These dermatoses usually disappear when contact with the sensitizer is eliminated.

PAHs have also been shown to cause cytotoxicity in rapidly proliferating cells throughout the body, with the hematopoietic system, lymphoid system, and testes frequently noted as targets (Santodonato et al., 1981). This effect appears to be due to inhibition of DNA replication by the PAHs. Destruction of the sebaceous glands, hyperkeratosis, hyperplasia, and ulceration have been observed in mouse skin following dermal application of the carcinogenic PAHs, with the degree of induced morphological changes appearing to correlate with the carcinogenic activity. However, it does not seem that this dermal toxicity is necessary or sufficient for carcinogenesis (Santodonato et al., 1981). It should be noted that similar types of dermatitis have been observed in workers exposed to such PAH-containing materials as coal tar and mineral oil. The carcinogenic PAHs have also been shown to have an immunosuppressive effect in animals. Again, it is not clear what relationship, if any, this immunosuppression has with carcinogenesis.

Some of the noncarcinogenic PAHs have been shown to cause systemic toxicity, but these effects are generally seen only at high doses (Santodonato et al., 1981). For example, slight morphological changes in the livers and kidneys of rats have been reported following oral adminstration of acenaphthene. Oral administration of naphthalene to rabbits has resulted in cataract formation.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

IARC (1983) in reviewing the carcinogenicity of the PAHs, indicated those for which there was sufficient, limited, inadequate, or adequate negative evidence of carcinogenicity (Table 1). The more potent carcinogens are almost certainly included within the group for which sufficient evidence of carcinogenicity is available. For purposes of risk assessment, it is necessary to consider that the potencies of different PAHs vary over a wide range and that a number of factors, including factors specific to the chemical, the host animal, and the circumstances of exposure, affect carcinogenic potency.

The approach adopted by EPA (1980a, 1984a) as the basis for risk assessment is to apply a carcinogenic potency factor calculated from benzo(a)pyrene assays to the subclass of carcinogenic PAHs within the particular mixture that is to be assessed. This approach involves three assumptions: (1) that all carcinogenic PAHs have the same potency as benzo(a)pyrene; (2) that their effects are additive; and (3) that the noncarcinogenic PAHs do not contribute to the carcinogenic effects of the mixture. Although there is limited empirical evidence to support assumptions (2) and (3), assumption (1) may lead to large overestimates of risk because benzo(a)pyrene is one of the most potent carcinogens among the PAHs and is usually present only in a small percentage of the total mixture. Benzo(a)pyrene is representative of the carcinogenic PAHs and is classified by EPA (1986) in Group B2--Potential Human Carcinogen.

EPA (1980a, 1984a) calculated a value of 11.5 $(mg/kg/day)^{-1}$ as the carcinogenic potency factor (upper bound on lifetime risk) for oral exposure to carcinogenic PAHs, based on the study of Neal and Rigdon (1967) in which oral administration of benzo(a)pyrene led to forestomach tumors in mice. EPA (1984a) calculated a cancer potency factor for inhalation of benzo(a)pyrene based on the study of Thyssen et al. (1981). This assay evaluated the production of respiratory tract tumors in hamsters using benzo(a)pyrene at concentrations of 2.2-9.5 mg/m^3. The linearized multistage model yielded a

carcinogenic potency factor of 6.11 $(mg/kg/day)^{-1}$. These cancer potency estimates are upper bound limits on potency; the actual potency is unlikely to be greater, but may be lower. These estimates are currently under review at EPA (ATSDR, 1987).

EPA's Carcinogen Assessment Group has not reported a risk assessment for dermal exposure to carcinogenic PAHs. Santodonato et al. (1981) performed risk assessments for both dermal and oral exposure and indicated that benzo(a)pyrene was more potent when applied dermally than when administered orally. A number of factors may account for this difference in relative potency and a complete derivation of a dermal potency factor is beyond the scope of this profile.

In addition to quantification of the effects of individual PAHs, EPA developed a cancer potency factor for inhalation of coal tar pitch volatiles (EPA, 1984b). This study evaluated epidemiological data from exposure of coke oven workers to between 0 and greater than 700 mg/m^3-month coal tar vapors. The equivalent incremental risk calculated from the study was 3.2 $(mg/kg/day)^{-1}$. EPA (1984b) classified coal tar pitch volatiles in Group A--Human Carcinogen.

Based on a study in which ocular lesions were observed in rats (Schmahl, 1955) and epidemiologic data on occupationally-exposed coke oven workers, EPA (1986) developed an oral RfD for chronic exposure to naphthalene, a noncarcinogenic PAH. An uncertainty factor of 100 was used to derive the RfD of 4.1×10^{-1} mg/kg/day.

SUMMARY OF PAHs CRITERIA

EPA Carcinogen Classification	
Benzo(a)pyrene	Group B2
Oral carcinogenic potency factor	
Benzo(a)pyrene	11.5 $(mg/kg/day)^{-1}$
Inhalation carcinogenic potency factor	
Benzo(a)pyrene	6.11 $(mg/kg/day)^{-1}$
RfD	
Naphthalene	0.41 mg/kg/day
MCL	Not Available
MCLG	Not Available

TABLE A-1

CLASSIFICATION OF PAHs ACCORDING TO
EVIDENCE FOR CARCINOGENICITY

Chemicals for which there is sufficient evidence that they are carcinogenic in animals:

Benzo(a)anthracene
Benzo(b)fluoranthene
Benzo(j)fluroanthene
Benzo(k)fluoranthene
Benzo(a)pyrene
Dibenzo(a,h)acridine
Dibenzo(a,j)acridine
Dibenzo(a,h)anthracene
7H-Dibenzo(c,g)carbazole
Dibenzo(a,e)pyrene
Dibenzo(a,h)pyrene
Dibenzo(a,i)pyrene
Dibenzo(a,l)pyrene
Indeno(1,2,3-c,d)pyrene
5-Methylchrysene

Chemicals for which there is limited evidence that they are carcinogenic in animals:

Anthranthrene
Benzo(c)acridine
Carbazole
Dibenzo(a,e)fluoranthene
Chrysene
Cyclopenta (c,d)pyrene
Methylfluoranthene
Dibenzo(a,c)anthracene
Dibenzo(a,j)anthracene
2-, 3-, 4-, and 6-Methylchrysene
2- and 3-

Chemicals for which the evidence is inadequate to assess their carcinogenicity:

Benzo(a)acridine
Benzo(g,h,i)fluoranthene
Benzo(a)fluroene
Benzo(b)fluroene
Benzo(c)fluroene
Benzo(g,h,i)perylene
Benzo(c)phenanthrene
Benzo(e)pyrene
Coronene
1,4-Dimethylphenanthrene
Fluorene
1-Methylchrysene
1-Methylphenanthrene
Perylene
Phenanthrene
Triphenylene

Chemicals for which the available data provide adequate negative evidence that they are carcinogenic:

Anthracene
Fluroanthene
Pyrene

SOURCE: IARC, 1983

REFERENCES

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987a. Draft Toxicological Profile for Benzo(a)anthracene. U.S. Public Health Service.

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987b. Draft Toxicological Profile for Benzo(b)fluoranthene. U.S. Public Health Service.

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987c. Draft Toxicological Profile for Benzo(a)pyrene. U.S. Public Health Service.

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987d. Draft Toxicological Profile for Dibenz(a,h)anthracene. U.S. Public Health Service.

DIPPLE, A., MOSCHEL, R.C., and BIGGER, C.A.H. 1984. Polynuclear aromatic carcinogens. In Searle, C.E. (ed.). Chemical Carcinogens-Second edition. ACS Monograph 182. American Chemical Society. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980a. Ambient Water Quality Criteria for Polynuclear Aromatic Hydrocarbons. Office of Water Regulations and Standards. October. Washington, D.C. EPA 44/5-80-069.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980b. Ambient Water Quality Criteria for Acenaphthene. Office of Water Regulations and Standards. October. Washington, D.C. EPA 440/5-80-015.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980c. Ambient Water Quality Criteria Document for Fluoranthene. Office of Water Regulations and Standards. October. Washington, D.C. EPA 440/5-80-049.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980d. Ambient Water Quality Criteria for Naphthalene. Office of Water Regulations and Standards. October. Washington, D.C. EPA 440/5-80-059.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984a. Health Effects Assessment for Polycyclic Aromatic Hydrocarbons (PAHs). Environmental Criteria and Assessment Office. September. Cincinnati, Ohio. EPA 540/1-86-013.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984b. Health Effects Assessment for Coal Tars. Environmental Criteria and Assessment Office. September. Cincinnati, Ohio. EPA 540/1-86-024.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984c. Health Effects Assessment for Benzo(a)pyrene. Environmental Criteria and Assessment Office,. September. Cincinnati, Ohio. EPA 540/1-86-022.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984d. Health Effects Assessment for Naphthalene. Environmental Criteria and Assessment Office. September. Cincinnati, Ohio. EPA 540/1-86-014.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984e. Health Effects Assessment for Phenanthrene. Environmental Criteria and Assessment Office. September. Cincinnati, Ohio. EPA 540/1-86-029.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984f. Health Effects Assessment for Pyrene. Environmental Criteria and Assessment Office. September. Cincinnati, Ohio. EPA 540/1-86-030.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. An exposure and Risk Assessment for Benzo [a]pyrene and Other Polycyclic Aromatic Hydrocarbons. Volume IV. Office of Water, Washington, D.C. EPA 440/4-85-020-V4.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Health and Environmental Effects Profile for Naphthalene. Environmental Criteria and Assessment Office, Cincinnati, OH.

FRIERSON, M.R., KLOPMAN, G., and ROSENKRANZ, H.S. 1986. Structure-activity relationships (SARs) among mutagens and carcinogens: A Review. Environ. Mutagen. 8:283-327.

HERBES, S.E, and SCHWALL, L.R. 1978. Microbial transformation of polycyclic aromatic hydrocarbons in pristine and petroleum contaminated sediments. App. Environ. Microbiol. 35:306-316.

HINCHEE, R.E., and REISINGER, H.J. 1987. A practical application of multiphase transport theory to groundwater contamination problems. GWMR, Winter 1987, p. 84-92.

HUEREN, K. 1983. Handbood of Environmental Data on Organic Chemicals. Van Nostrand Reinhold Co., New York.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1973. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Volume 3. Certain Polycyclic Aromatic Hydrocarbons and Heterocyclic Compounds. World Health Organization, Lyon, France.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1983. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol. 32: Polynuclear Aromatic Compounds; Part 1, Chemical, Environmental, and Experimental Data. World Health Organization, Lyon, France.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1984. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 33: Polynuclear Aromatic Compounds, Part 2, Carbon Blacks, Mineral Oils and Some Nitroarenes. World Health Organization, Lyon, France.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1985. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 35: Polynuclear Aromatic Compounds, Part 4, Bitumens, Coal-tar and Derived Products, Shale-oils, and Soot. World Health Organization, Lyon, France.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1977. Criteria for a Recommended Standard-Occupational Exposure to Coal Tar Products. Washington, D.C. DHEW (NIOSH) 78-107.

NEAL, J., and RIDGON, R.H. 1967. Gastric tumors in mice fed benzo(a)pyrene: A quantitative study. Tex. Rep. Biol. Med. 25:553-557.

RADDING, S.B., LIU, D.J., JOHNSON, H.L., MILL, T. 1976. The Environmental Fate of Selected Polynuclear Aromatic Hydrocarbons. EPA 560/5-65-009.

RIGDON, R., and NEAL, J. 1969. Relationship of leukemia to lung and stomach tumors in mice fed benzo(a)pyrene. Proc. Soc. Exp. Biol. Med. 130:146-148.

SANTODONATO, J., HOWARD, P., and BASU, D. 1981. Health and ecological assessment of polynuclear aromatic hydrocarbons. J. Environ. Pathol. Toxicol. 5:1-364.

TABAK, H.H., QUAVE, S.A., MASHNI, C.I, and BARTH, E.F. 1981. Biodegradability studies with organic priority pollutant compounds. J. Water. Pollut. Control Fed. 53:1503-1518.

THYSSEN, J., ALTHOFF, J., KIMMERLE, G., and MOHR, U. 1981. Inhalation studies with benzo(a)pyrene in Syrian golden hamsters. J. Natl. Cancer Int. 66:575-577.

VEITH, G.D., DEFOE, D., and KNUTH, M., 1985. Structure-activity relationships for screening organic chemicals for potential ecotoxicity effects. Drug Metabolism Reviews 15(7): 1295-1303.

A.8 TETRACHLOROETHYLENE

INTRODUCTION

Tetrachloroethylene is a moderately volatile liquid with important uses in commercial dry cleaning operations. It is also used in the textile industry, in the synthesis of certain fluorocarbons, and as a degreaser. It is a common contaminant in air, groundwater, and surface water (EPA, 1987a). Mean ambient air concentrations in the U.S. have been estimated as 160 ppt in rural areas, 790 ppt in urban and suburban areas and 1300 ppt in areas near emission sources (Brodzinsky and Singh, 1982). EPA (1985a) estimated worldwide average ambient air concentrations of 40 ppt in the northern hemisphere and 12 ppt in the southern hemisphere. Tetrachloroethylene has been found in drinking water throughout the U.S. with mean levels ranging from 0.01 - 30 μg/liter (ATSDR, 1987).

TOXICOKINETICS

Tetrachloroethylene is readily absorbed through the lungs (Monster et al., 1979) and gastrointestinal tract (Frantz and Watanabe, 1983). Tetrachloroethylene vapors and liquid are poorly absorbed through the skin (Riihimaki and Pflaffli, 1978; EPA, 1985a,b). Once absorbed, tetrachloroethylene is distributed primarily to adipose tissue and other lipid-rich tissues (Savolainen et al., 1977). Excretion of tetrachloroethylene is primarily by exhalation of the unmetabolized compound. Metabolism by the liver does occur, but accounts for only a small proportion of an absorbed dose (Monster et al., 1979; Frantz and Watanabe, 1983). The metabolic pathways appear to be saturable at air concentrations >100 ppm (Ohtsuki et al., 1983). Metabolite excretion is through the urine (ATSDR, 1987).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

CARCINOGENIC POTENTIAL

Epidemiological studies of dry cleaning workers suggest that chronic occupational exposure to tetrachloroethylene may be associated with increased cancer risks. However, the evidence is inconclusive due to concomitant worker exposures to other chemicals and to the occurrence of other potentially confounding factors such as smoking and socioeconomic status not addressed in these studies (ATSDR 1987).

In a National Cancer Institute bioassay (1977), a high incidence of hepatocellular carcinoma was observed in both sexes of $B6C3F^1$ mice administered tetrachloroethylene in corn oil by gavage 5 days per week for 78 weeks and observed for an additional period of 32 weeks (rats) or 12 weeks (mice). Time-weighted average doses were 536 and 1,072 mg/kg/day in males and 386 and 772 mg/kg/day in females. No conclusion concerning the effects on Osborne-Mendel rats administered 471 to 949 mg/kg by gavage could be made because of high mortality rates and other technical problems with the study (NCI, 1977).

The National Toxicology Program (NTP) recently reported that tetrachloroethylene is carcinogenic in inhalation studies (NTP, 1986). In the NTP study, F344/N rats were exposed to 0, 200 or 400 ppm for 103 weeks. Increased incidences of mononuclear cell leukemia were seen in both sexes of rats and increased dose-related incidences of renal adenomas and carcinomas (combined) were seen in males only. Significantly increased dose related incidences of hepatocellular carcinomas were observed in mice of both sexes exposed to 0, 100 or 200 ppm for 103 weeks. Classification of tetrachloroethylene as a carcinogen in the rat is controversial because of questions raised concerning the relevance to humans of increased incidences of mononuclear cell leukemia and the validity of combining renal adenomas and carcinomas to achieve statistical significance (EPA, 1987a).

GENOTOXIC POTENTIAL

The majority of *in vivo* and *in vitro* genotoxicity studies, using a variety of test systems, revealed little or no evidence of activity by tetrachloroethylene (ATSDR, 1987; EPA, 1985a,b). Cerna and Kypenova (1977, abstract only) reported positive results in plate tests with *S. typhimurium* and in host-mediated assays (EPA, 1985a,b). Price et al. (1978) reported positive results for cell transformation in rat embryo cells. Beliles et al. (1980) reported mixed results indicating DNA damaging potential in human fibroblasts as measured by unscheduled DNA synthesis. A positive response was seen at the lowest dose tested, but negative responses were seen at two higher doses (EPA, 1985b). Walles et al. (1986) reported positive results for induction of single strand breaks in mouse DNA.

REPRODUCTIVE EFFECTS

Fetotoxic effects were observed in the offspring of female rats and mice exposed to tetrachloroethylene at 300 ppm for 7 hours daily on days 6-15 of gestation, including a decrease in fetal body weight in mice and a small but significant increase in fetal resorption in rats. The pregnant mice had significantly increased relative liver weights, and their pups exhibited developmental effects, including subcutaneous edema and delayed ossification of skull bones and sternebrae (Schwetz et al., 1975).

ACUTE/CHRONIC EFFECTS

Tetrachloroethylene is not highly toxic in humans. In mice, oral LD_{50} values for tetrachloroethylene range from 6.4 - 10.8 g/kg, (IARC, 1979). In rats, oral LD_{50}'s of 3 - 4 g/kg (Hayes et al., 1986) and 13g/kg (Smyth et al., 1969) have been reported. These LD_{50} values suggest that tetrachloroethylene is a slightly toxic to practically non-toxic compound (Hodge and Sterner, 1949). The inhalation LC_{50} values in mice and rats with 4-hour inhalation exposures have been reported to be 5,200 ppm (Friberg et al., 1953) and 4,000 ppm (Carpenter et al., 1949) respectively.

The principal toxic effects of tetrachloroethylene in humans and animals from both acute and longer-term exposures include central nervous system (CNS) depression and fatty infiltration of the liver and kidney with concomitant changes in serum enzyme levels indicative of tissue damage (EPA, 1985a,b).

Central Nervous System Effects

Individuals exposed to tetrachloroethylene concentrations ranging from 900 - 1500 ppm experienced lassitude, mental fogginess or exhilaration, progressing at the higher dose to signs of inebriation (EPA, 1980). Exposure to lower concentrations, 20 - 150 ppm for 5 weeks resulted in decreased odor perception and diminished response on a neurological test at 100 ppm but not at 20 ppm (Stewart et al., 1974). Signs of central nervous system depression and cholinergic stimulation were also observed at concentrations of 235 ppm in an animal study involving rabbits, monkeys, rats, and guinea pigs (EPA, 1980). ATSDR (1987) concluded that the threshold for CNS effects from acute exposure in humans may be in the 100 - 200 ppm range and that the results of several animal studies are not inconsistent with this range.

Liver And Kidney Effects

Hepatotoxic effects reported in humans exposed to tetrachloroethylene include cirrhosis, toxic hepatitis, liver cell necrosis, hepatomegaly and altered liver function (EPA, 1985a). Three of seven men occupationally exposed to tetrachloroethylene at concentrations of 300 - 400 ppm showed evidence of impaired liver function (EPA, 1980).

Single oral gavage doses of 2,158 mg/kg tetrachloroethylene given to rabbits resulted in an increase in serum lipoprotein levels and serum enzymes indicative of liver damage (Fuji, 1975). Increased total lipid and triglyceride levels were observed in mice exposed to 800 ppm tetrachloroethylene in air for 3 hours (Ogata et al., 1968). A dose-related

increase in fatty infiltration of the livers of mice was observed following 4-hour inhalation exposures to 200 and 3,000 ppm (EPA, 1985a,b).

Rats exposed to 1,600 ppm tetrachloroethylene 7 hours per day, 5 days per week, 18 times over 25 days exhibited central nervous system depression and hepatic and renal hypertrophy. Rats exposed to 230 ppm and 470 ppm tetrachloroethylene, 8 hours per day, 5 days per week, over a period of 7 months, exhibited congestion and swelling of kidneys and liver, respectively (Carpenter, 1937). Female Sprague-Dawley rats exposed to tetrachloroethylene in air 5 days a week for 12 months at concentrations of 300 or 600 ppm showed liver atrophy, and the high-dose females developed an increased incidence of fluid-filled cysts in the liver (EPA, 1980).

Fatty infiltration in the livers of mice was observed following exposure to 200 ppm, 4 hours per day, 5 days per week for 8 months (Kylin et al., 1965). A high incidence of toxic nephropathy was seen in mice and rats exposed orally to 500 mg/kg tetrachloroethylene for 78 weeks (NCI, 1977).

In guinea pigs, a dose-dependent increase in liver weight and fatty infiltration of the liver was observed following exposure to 100, 200, or 400 ppm tetrachloroethylene for up to 169 exposures over 236 days (Rowe et al., 1952).

Rabbits showed liver enzyme changes and renal function alterations following exposure to 200 - 300 ppm, 4 hours per day, 5 days per week for 9 weeks (Brancaccio et al., 1971; Mazza, 1972).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

EPA's Carcinogen Assessment Group has classified tetrachloroethylene in Group B2—Probable Human Carcinogen based on inconclusive evidence for carcinogenicity in humans and sufficient evidence for carcinogenicity in animals (EPA, 1986b). This classification has been questioned by the Science

Advisory Board, Halogenated Organics Subcommittee, which has recommended classification in Group C-Possible Human Carcinogen (EPA, 1987a).

In its Health Assessment Document for tetrachloroethylene, EPA (1985a) reported a carcinogenic potency factor for exposure to this compound by ingestion. The EPA Carcinogen Assessment Group (CAG) derived the oral carcinogenic potency factor based on the results of the NCI (1977) gavage bioassay for mice cited above. Groups of 50 male and 50 female B6C3F_1 mice were exposed to tetrachloroethylene in corn oil by gavage, 5 days per week, for 78 weeks. The time-weighted average doses for this study were 536 or 1,072 mg/kg/day for male mice, and 386 or 772 mg/kg/day for female mice. Control groups of 20 male and 20 female mice were also maintained. All surviving mice were killed at 90 weeks. Highly significant increases in the incidences of hepatocellular carcinomas were observed in treated mice. The incidences for these tumors were 2/20, 32/48, and 27/45 for the control, low-dose, and high-dose male mice, respectively, and 0/20, 19/48, and 19/45 for the control, low-dose, and high-dose female mice, respectively. The dose-response data for female mice were determined to be more reliable, and were therefore used to calculate a carcinogenic potency. Using the linearized multistage model and appropriate scaling factors, an oral carcinogenic potency factor of 5.1×10^{-2} $(mg/kg/day)^{-1}$ was derived. This value represents the 95% upper bound limit of the carcinogenic potency for tetrachloroethylene, the true value may be lower than this estimate but is unlikely to be greater.

An inhalation carcinogenic potency factor was also estimated from the hepatocellular carcinoma data for female mice in the NCI (1977) bioassay; however this value has recently been reevaluated on the basis of the NTP (1986) inhalation study. EPA (1986a) used six sets of incidence data from this study to calculate new cancer potency factors, ranging from $1.0 - 3.3 \times 10^{-3}$ $(mg/kg/day)^{-1}$. The tumor incidence data on which these calculations were based were for mononuclear cell leukemia in male and female rats, liver carcinomas in male and female mice, and combined incidences of liver adenomas and carcinomas in male and female mice. An inter-office EPA work group is currently reviewing the cancer potency factor for tetrachloroethylene (EPA,

1988). Consequently no values are presently in EPA's Integrated Risk Information System regarding the carcinogenic potential of tetrachloroethylene.

EPA derived an oral reference dose (RfD) of 1×10^{-2} mg/kg/day for tetrachloroethylene (EPA, 1988) based on a study in mice. In this study (Buben and O'Flaherty, 1985) mice were exposed to tetrachloroethylene in corn oil gavage for 5 days/week for 6 weeks and a no-observed-adverse effect level (NOAEL) of 14 mg/kg/day was identified for hepatotoxic effects.

The EPA Office of Drinking Water developed ten-day, longer-term, and lifetime health advisories (HAs) (EPA, 1987a). The ten-day HA derived for a 10-kg child is 2,000 μg/liter. This HA is based on a study by Buben and O'Flaherty (1985) in which mice were treated by gavage with doses ranging from 20 to 2,000 mg/kg body weight, 5 days/week for 6 weeks. A no-observed-adverse effect level (NOAEL) of 20 mg/kg body weight was identified based on significant increases in liver weight at higher doses. Longer-term HAs were also derived from the NOAEL identified in this study. A longer-term HA was derived for a 10-kg child consuming 1 liter of water per day and a 70-kg adult consuming 2 liters of water per day. These values are 1,400 μg/liter and 5,000 μg/liter, respectively.

EPA (1987a) also derived a lifetime drinking water equivalent level (DWEL) for tetrachloroethylene. A DWEL is defined as the medium-specific concentration (e.g., in water) which is considered to be protective for noncarcinogenic toxic endpoints over a lifetime of exposure. A DWEL of 500 μg/liter was determined for a 70-kg adult ingesting 2 liters of water per day, based on the NOAEL of 20 mg/kg body weight established in the study cited above. The lifetime HA of 10 μg/liter is derived from the DWEL using a relative source contribution of 20% and an uncertainty factor of 10. This value can be converted to an oral reference dose (RfD) of 0.0143 μg/kg/day, which corresponds well with the verified RfD of 0.01 mg/kg/day presented by EPA (1987b).

SUMMARY OF TETRACHLOROETHYLENE CRITERIA

EPA carcinogen classification	Group B2
Oral carcinogenic potency factor	5.1×10^{-2} $(mg/kg/day)^{-1}$
Inhalation carcinogenic potency factor $(mg/kg/day)^{-1}$	$1.0\text{-}3.3 \times 10^{-3}$
RfD, verified	0.01 mg/kg/day
MCL	Not Available
MCLG	Not Available
Health Advisories (HA):	
Ten-Day HA (child)	2,000 µg/liter
Longer-term HA	
Child	1,400 µg/liter
Adult	5,000 µg/liter
Lifetime HA (DWEL)	500 µg/liter

REFERENCES

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987. Draft Toxicological Profile for Tetrachloroethylene; U.S. Public Health Service 1987

ATKINSON, R. 1985. Kinetics and mechanisms of the gas-phase reactions of the hydroxyl radical with organic compounds under atmospheric conditions. Chem. Rev. 85:130-132 (As cited in ATSDR, 1988)

BELILES, R.P., BURSISK, D.J., and MECLER, F.J. 1980. Teratogenic, mutagenic risk of workplace contaminants: Trichloroethylene, perchloroethylene, and carbon disulfide. Unpublished studies. U.S. Dept of Health, Education, and Welfare. Contract No. 210-77-0047. Litton Bionetics, Inc., Kensington, MD. (Cited in EPA, 1985a)

BOUWER, E.J. and McCARTY, P.L. 1983. Transformations of 1- and 2- carbon halogenated aliphatic organic compounds under methanogenic conditions. Appl. Environ. Microbiol. 45(4):1286-1294

BRANCACCIO, A., MAZZA, V., and DIPAOLO, R. 1971. Renal function in experimental tetrachloroethylene poisoning. Folia Med. (Naples). 54:233-237 (As cited in EPA, 1987a).

BRODZINSKY, R. and SINGH, H.B. 1982. Volatile organic chemicals in the atmosphere. An assessment of available data. Menlo Park, CA: Atmospheric Science Center, SRI International. Contract 68-02-3452; 109-110. (As cited in ASTDR, 1987)

BUBEN, J.A. and O'FLAHERTY, E. 1985. Delineation of the role of metabolism in the hepatotoxicity of trichloroethylene and perchloroethylene: a dose-effect study. Toxicol. Appl. Pharmacol. 78:105-122 (As cited in EPA, 1987a and EPA, 1988)

CARPENTER, D.P. 1937. The chronic toxicity of tetrachloroethylene. J. Ind. Hyg. Toxicol; 19:323-336. (Cited in EPA, 1985a,b)

CARPENTER, C.P., SMYTH, H.F, and POZZANI, U.C. 1949. The assay of acute vapor toxicity and the grading and interpretation of results on 96 chemical compounds. J. Ind. Hyg. Toxicol.; 31:343-346. (As cited in EPA, 1987a).

CERNA, M. and KYPENOVA, H. 1977. Mutagenic activity of chloroethylenes analyzed by screening system tests. Mutat. Res. 46:214-215

DILLING, W.L., TEFERTILLER, N.B., and KALLOS, G.J. 1975. Evaporation rates of methylene chloride, chloroform, 1,1,1-trichloroethane, trichloroethylene, tetrachloroethylene, and other chlorinated compounds in dilute aqueous solutions. Environ. Sci. Technol. 9(9):833-838 (As cited in EPA, 1979)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Washington, D.C. December 1979. EPA 440/4-79-029

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Tetrachloroethylene. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-073

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Health Assessment Document for Tetrachloroethylene (Perchloroethylene). Office of Health and Environmental Assessment, Washington, D.C. July 1985. EPA 600/8-82-005F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. Drinking Water Criteria Document for Tetrachloroethylene. Office of Drinking Water, Criteria and Standards Division, Washington, D.C. April 1985

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986a. Addendum to the Health Assessment Document for Tetrachloroethylene (perchloroethylene). External Review Draft. Office of Health and Environmental Assessment. April 1986

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986b. Guidelines for carcinogen risk assessment. Fed. Reg. 51:3392-34003 (September 24, 1986)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986c. Superfund Public Health Evaluation Manual. Office of Emergency and Remedial Response. EPA 540/1-86/060

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987a. Health Advisory for Tetrachloroethylene. Office of Drinking Water, Washington, D.C. March 31, 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987b. Updated Reference Dose and Cancer Potency Numbers for Use in Risk Assessments. Memorandum from Sandra Lee, Toxics Integration Branch, Washington D.C. November 16, 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Integrated Risk Information System (IRIS). On-line. 1989.

FRANTZ, S.W. and WATANABE, P.B. 1983. Tetrachloroethylene: Balance and tissue distribution in male Sprague-Dawley rats by drinking water administration. Toxicol. Appl. Pharmacol.; 69:66-72.

FREIBERG, L., KYLIN, B., and NYSTROM, A. 1953. Toxicities of trichloroethylene and tetrachloroethylene and Fujiwara's pyridine-alkali reaction. Acta Pharmacol. Toxicol.; 9:303-312 (As cited in ATSDR, 1987).

FUJI, T. 1975. The variation in the liver function of rabbits after administration of chlorinated hydrocarbons. Jap. J. Ind. Health; 17:81-88. (As cited in EPA, 1987a)

HAYES, J.R., CONDIE, L.W., and BORZELLECA, J.F. 1986. The subchronic toxicity of tetrachloroethylene (perchloroethylene) administered in the drinking water of rats. Fund. Appl. Toxicol; 7(1):119-125. (As cited in ATSDR, 1987)

HODGE, H.C. and STERNER, J.H. 1949. Tabulation of Toxicity Classes. Am. Ind. Hyg. Assoc. Q. 10:93-96

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1979. IARC Monographs on the Evaluation of the Carcinogenic Risks of Chemicals to Humans. Vol. 20: Some Halogenated Hydrocarbons. World Health Organization, Lyon, France

KEIL, S.L. 1979. Tetrachloroethylene. In: Kirk-Othmer Concise Encyclopedia of Chemical Technology, Vol. 5 Grayson, M. and Eckroth, P. eds. New York: John Wiley and Sons, Inc. pp. 754-762 (As cited in ATSDR, 1987)

KYLIN, B., SUMEGI, I., and YLLNER, S. 1965. Hepatotoxicity of inhaled trichloroethylene and tetrachloroethylene - long-term exposure. Acta Pharmacol. Toxicol.; 22:379-385 (As cited in ATSDR, 1987).

LAMBERT, S.M. 1933. No title provided. JAMA; 100:247. (As cited in ATSDR, 1987)

MAZZA, V. 1972. Enzyme changes in experimental tetrachloroethylene intoxication. Folia Med. 55(9-10):373-381 (As cited in EPA, 1987a).

MONSTER, A.C., BOERSMA, G., and STEENWEG, H. 1979. Kinetics of tetrachloroethylene in volunteers: Influence of exposure concentration and work load. Int. Arch. Occup. Environ. Health; 42:303-309 (As cited in ATSDR, 1987).

NATIONAL CANCER INSTITUTE (NCI). 1977. Bioassay of Tetrachloroethylene for Possible Carcinogenicity. CAS No. 127-18-4. NCI Carcinogenesis Technical Report Series No. 13. Washington, D.C. DHEW (NIH) Publication No. 77-813

NATIONAL TOXICOLOGY PROGRAM (NTP). 1986. Toxicology and Carcinogenesis Studies of Tetrachloroethylene (Perchloroethylene) (CAS No. 127-18-4) in F344/N Rats and B6C3F_1 Mice (Inhalation Studies). NTP Technical Report Series No. 311. Research Triangle Park, North Carolina. DHEW (NIH) Publication No. 86-2567

NEELY, W.B., BRANSON, D.R., and BLAU, G.E. 1974. Partition coefficients to measure bioconcentration potential of organic chemicals in fish. Environ. Sci. Technol. 8: 1113-1115 (As cited in EPA, 1979)

OGATA, M., TOMOKUNI, K., and WATANABE, S. 1968. ATP and lipid contents in the liver of mice after inhalation of chlorinated hydrocarbons. Ind. Health; 6:116-119. (As cited in EPA, 1987a).

OHTSUKI, T., SATO, K., KOIZUMI, A., KUMAI, M., and IKEDA, M. 1983. Limited capacity of humans to metabolize tetrachloroethylene. Int. Arch. Occup. Environ. Health; 51:381-390. (As cited in ATSDR, 1987).

PRICE, P.J., WASSETT, C.M., and MANSFIELD, J.I. 1978. Transforming activities of trichloroethylene and proposed industrial alternatives. In Vitro 14(3):290-293 (As cited in ATSDR, 1987).

RIIHIMAKI, V. and PFAFFLI, P. 1978. Percutaneous absorption of solvent vapors in man. Scand. J. Work Environ. Health; 4: 73-85 (As cited in ATSDR, 1987).

ROWE, V.K., McCOLLISTER, D.D., SPENCER, H.C., ADAMS, E.M., and IRISH, D.D. 1952. Vapor toxicity of tetrachloroethylene for laboratory animals and human subjects. AMA Arch. Ind. Hyg. Occup. Med.; 5:566-579 (As cited in ATSDR, 1987).

SAVOLAINEN, H., PFAFFLI, P., TENGEN, M., and VAINIO, H. 1977. Biochemical and behavioral effects of inhalation exposure to tetrachloroethylene and dichloromethane. J. Neuropath. Exp. Neurology; 36:941-949 (As cited in ATSDR, 1987).

SCHWETZ, B.A., LEONG, B.K.J., and GEHRING, P.J. 1975. The effect of maternally inhaled trichloroethylene, perchloroethylene, methylchoroform, and methylene chloride on embryonal and fetal development in mice and rats. Toxicol. Appl. Pharmacol.; 32:84-96. (As cited in ATSDR, 1987).

STEWART, R.D., HAKE, C.L., FORSTER, H.V., LEBRUN, A.J., PETERSON, J.F., and WU, A. 1974. Tetrachloroethylene: Development of a biologic standard for the industrial worker by breath analysis. Report No. NIOSH-MCOW-ENUM-PCE-74-6, Medical College of Wisconsin, Milwaukee, Wisconsin (As cited in EPA, 1987a).

VERSCHUEREN, K. 1983. Handbook of Environmental Data on Organic Chemicals. Van Nostrand Reinhold Co., New York.

VOGEL, T.M. and McCARTY, P.L. 1985. Biotransformation of tetrachloroethylene to trichloroethylene, dichloroethylene, vinyl chloride, and carbon dioxide under methanogenic conditions. Appl. Environ. Microbiol. 49(5): 1080-1083

WALLES, S.A.S. 1986. Induction of single-strand breaks in DNA of mice by trichloroethylene and tetrachloroethylene. Toxicol. Lett. 31(1):31-35 (As cited in ATSDR, 1987).

WEAST, R.E., ed. 1983. Handbook of Chemistry and Physics. 63rd ed. CRC Press, Cleveland, Ohio.

WILSON, J.T., ENFIELD, C.G., DUNLAP, W.J., COSBY, R.L., FOSTER, D.A., and BASKIN, L.B. 1981. Transport and fate of selected organic pollutants in a sandy soil. J. Environ. Qual. 10(4):501-506

A.9 TRICHLOROETHYLENE

INTRODUCTION

Trichloroethylene is a colorless, highly volatile liquid widely used as a metal degreaser. It is a common contaminant in air, soil, groundwater, and surface water (EPA, 1985a). U.S. mean ambient air concentrations of 30 ppt in rural areas and 460 ppt in urban areas have been reported (Brodzinsky and Singh, 1982). EPA (1985a) has also reported average trichloroethylene levels of 11-30 ppt in the northern hemisphere and <3 ppt in the southern hemisphere. Trichloroethylene was detected in one national survey of U.S. drinking waters at a mean concentration of 2.1 μg/liter. Other water surveys reporting median concentrations ranging from 0.01 to approximately 1 μg/liter have been reviewed by ATSDR (1988).

TOXICOKINETICS

The toxicokinetics and metabolism of trichloroethylene have been studied in humans as well as in laboratory animals. Following inhalation exposure, absorption in humans is proportional to concentration and duration of exposure. In mice and rats, absorption after oral ingestion is rapid and virtually complete (Prout et al., 1985). Liquid trichloroethylene can be absorbed through the skin in humans (Sato and Nakajima, 1978) and animals (Tsuruta, 1978), but dermal absorption of trichloroethylene vapor is negligible. Trichloroethylene is stored primarily in body fat (Savolainen et al., 1977). Metabolism occurs in the liver. Elimination is by urinary excretion of polar metabolites and by pulmonary excretion of unmetabolized trichloroethylene (EPA, 1985a).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

CARCINOGENIC POTENTIAL

EPA (1985a) and ATSDR (1988) have reviewed the carcinogenicity of trichloroethylene. Epidemiological studies of occupationally-exposed workers have been conducted and the majority of these tests have been negative (Axelson et al., 1978; Tola et al., 1978). Two studies reported increased tumor incidences (Barret et al., 1985; Blair et al., 1979), but in both cases exposure was to complex mixtures containing other potential carcinogens as well as trichloroethylene. In a review document, EPA (1985a) concluded that the epidemiologic data reviewed are inadequate for evaluating the carcinogenicity of trichloroethylene. Since the time of the EPA review, Axelson (1986) reported slight increases in the incidence of bladder cancer and lymphomas in exposed workers.

Several studies investigating the carcinogenic potential of trichloroethylene in animals have been conducted with mixed results. Trichloroethylene was negative following inhalation exposure to rats, mice and hamsters (Henschler et al., 1980) and oral exposure to mice (Henschler et al., 1984). Several studies in rats were judged to be inadequate for assessing carcinogenicity due to poor survival of the test animals (NCI, 1976; NTP, 1984/7).

Positive results have been obtained in one study in rats and several studies in mice. In male Sprague-Dawley rats, inhalation exposure resulted in increased incidences of testicular Leydig cell tumors and slight increases in leukemia and renal adenocarcinomas, and gavage exposure resulted in an increased incidence of leukemia (Maltoni et al., 1986). Two studies in mice, one using technical-grade trichloroethylene containing epichlorohydrin and the second using epichlorohydrin-free trichloroethylene, revealed significant increases in the incidence of liver tumors among both sexes of $B6C3F_1$ mice exposed by gavage (NCI, 1976; NTP, 1984). A third study indicated an increased incidence of pulmonary adenocarcinomas in female ICR mice after

inhalation exposure (Fukuda et al., 1983). Increased incidences of both pulmonary and liver tumors were also reported in $B6C3F_1$ and Swiss mice after inhalation exposure in a more recent study (Maltoni et al., 1986).

There is disagreement in the scientific community about the relevance of mouse liver tumors to human cancer risk. Several strains of laboratory mice, including the $B6C3F_1$ hybrid, appear to develop a high and variable proportion of liver tumors in one or both sexes with or without exposure to carcinogenic chemicals. Some scientists believe that the increased incidence of such tumors in animals treated with potential carcinogens such as trichloroethylene should be treated in the same manner as the increased incidence of tumors at other rodent organ sites, while others believe that mouse liver tumors are an experimental artifact which is not relevant to human hazard (EPA, 1985b).

GENOTOXIC POTENTIAL

Both positive and negative results have been reported from in vivo and in vitro genotoxicity assays. Positive results reported in human systems include tests for unscheduled DNA synthesis in human lymphocytes in vitro (Perocco and Prodi, 1981) and sister chromatid exchanges in circulating lymphocytes in workers, though the concentrations to which workers were exposed were not specified (Gu et al., 1981). Positive results have also been reported in in vitro assays for mutations and gene conversion in yeast (Bronzetti et al., 1978; Callen et al., 1980; Crebelli et al., 1985). Weakly positive results have been reported in bacterial gene mutation assays (Baden et al., 1979; Greim et al., 1975) and in transformation assays using mammalian cells (Price et al., 1978; Tu et al., 1985). In vivo, trichloroethylene was found to damage DNA in the livers of rats (Nelson and Bull, 1986) and mice (Walles, 1986), and induce gene mutations in yeast in a host-mediated assay (Bronzetti et al., 1978). ATSDR (1988) concluded that the evidence from these positive tests combined with negative results from other assays suggests, but does not prove, that trichloroethylene is genotoxic. EPA (1985a) also concluded that it may be a weakly active mutagen.

REPRODUCTIVE EFFECTS

Teratogenic effects have not been observed in the offspring of rats or mice exposed to 500 ppm trichloroethylene in air (Hardin et al., 1981). External hydrocephalus was observed in the offspring of rabbits similarly exposed. The authors concluded that this result could not be discounted due to the rarity of the anomaly; however, EPA (1985a) regarded the result as inconclusive. Fetotoxic effects including increased numbers of resorptions and reduced fetal body weight and delays in skeletal ossification have been observed in Wistar rats exposed to 100 ppm trichloroethylene in air during gestation (Healy et al., 1982). Similar effects were not seen in Sprague-Dawley rats or Swiss-Webster mice exposed to 300 ppm in air (Schwetz et al., 1975) or in Long-Evans rats exposed to 1800 ppm (Dorfmueller et al., 1979).

Reproductive tract effects observed in adult male mice include significant increases in sperm morphological abnormalities in mice exposed to 2000 ppm in air (Land et al., 1979) and decreases in sperm motility in mice orally exposed to a dose of approximately 780 mg/kg/day (EPA, 1985a).

ACUTE/CHRONIC EFFECTS

Systemic toxic effects of trichloroethylene have been reviewed by ATSDR (1988) and EPA (1985a). The principal target organs are the central nervous system (CNS), liver, kidney and hematological system.

In humans, the primary effects of short-term and chronic exposure involve CNS disturbances. Concentrations producing symptoms have not been precisely quantified. Short-term exposure to high concentrations has been reported to produce dizziness, headache, nausea, confusion, numbness and blurred vision (EPA, 1985a). Subjects exposed to 27, 81 to 201 ppm trichloroethylene in air for four hours reported drowsiness at 27 ppm, headache at 81 ppm and dizziness and anorexia at 201 ppm (Nomiyama and Nomiyama, 1977). Chronic exposure has been associated with more severe CNS

effects including ataxia, decreased appetite and sleep disturbances (EPA, 1985a). Workers exposed to 85 ppm trichloroethylene in air for an average of 3.75 years reported vertigo, fatigue, and headache more frequently than workers exposed to 13 or 34 ppm (Grandjean et al., 1955). However, this report cannot be considered conclusive because of uncertainties regarding concentration levels and the lack of a control group (ATSDR, 1988). Liver and kidney dysfunction associated with trichloroethylene exposure have been infrequently reported in humans (EPA, 1985a).

Industrial use of trichloroethylene is associated with dermatological effects including reddening, skin burns and dermatitis on contact. These effects are usually the result of contact with concentrated solvent, however, and no effects have been reported after exposure to trichloroethylene in dilute, aqueous solutions (EPA, 1985a).

In animals, inhalation of trichloroethylene has been reported to produce CNS effects, primarily behavioral changes, at concentrations above 100 ppm (ATSDR, 1988). Hepatotoxic and renal effects have been observed at lower doses. Liver enlargement is the most commonly observed hepatic effect, and mice appear to be more sensitive to hepatotoxicity than other species (Kjellstrand et al., 1981). NMRI mice exposed to 0, 37, 75, 150 or 300 ppm trichloroethylene in air for 3 weeks had liver weights that increased significantly in a dose-related manner (Kjellstrand et al., 1983). Histological alterations in liver cells were observed in $B6C3F_1$ mice exposed by gavage to 0, 250, 500, 1200 or 2400 mg/kg doses and increased liver weights were observed at 500 mg/kg and above (Stott et al., 1982). Rats exposed to 1100 mg/kg in the same study had increased liver weights, but no signs of hepatocellular alterations after three weeks of exposure.

Renal effects have been reported in animals following acute or chronic inhalation or oral exposure. Increased kidney weights were observed in NMRI mice exposed to air concentrations greater than or equal to 75 ppm for three weeks (Kjellstrand et al., 1983). Renal dysfunction has also been reported in rats exposed to 50, 200 or 800 ppm for 12 weeks (Nomiyama et al., 1986).

Chronic inhalation or oral exposure has produced histological renal tubular alterations and/or toxic nephropathy in several strains of rats (Maltoni et al., 1986; NCI, 1976; NTP, 1984, 1987) and in B6C3F_1 mice (NTP, 1984).

Hematological effects have been observed in rats following inhalation exposure. Continuous exposure to air concentrations equal to or above 50 ppm for 10 days produced dose-related decreases in enzyme activity in bone marrow and liver cells in rats (Fujita et al., 1984). Oral exposure to a dose of 437 mg/kg/day in drinking water produced decreased erythrocyte counts in male mice after four months exposure. Decreased leukocyte counts and increased fibrinogen were also reported in males and a shortened prothrombin time was reported in female mice (Tucker et al., 1982).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

EPA's Carcinogen Assessment Group (CAG) classified trichloroethylene in Group B2—Probable Human Carcinogen on the basis of sufficient animal evidence of carcinogenicity and inadequate human evidence (EPA, 1985a). This classification was affirmed in a more recent EPA (1987a) carcinogenicity assessment. The National Academy of Sciences has classified trichloroethylene as an animal carcinogen (EPA, 1985b). The International Agency for Research on Cancer (IARC) classified trichloroethylene in Category 3 (compound cannot be classified as to its carcinogenicity in humans).

EPA's Science Advisory Board critiqued the IARC classification. The majority of the Committee members classified the compound in IARC Category 3, while one member classified it in IARC category 2B (probable human carcinogen). The committee concluded that a definitive conclusion as to the classification of trichloroethylene could not be made because the interpretation of male mouse hepatocellular carcinomas is uncertain and the animal evidence is limited (EPA, 1985b).

EPA (1985a) calculated a cancer potency factor for oral exposure to trichloroethylene based on the incidences of liver hepatocellular carcinomas

in male and female mice exposed by gavage in two studies (NCI, 1976; NTP, 1984). These two studies are of adequate sample size and provide similar quantitative results. Separate cancer potency factors were calculated for each of the four sets of incidence data and the overall potency factor of 1.1 $\times 10^{-2}$ $(mg/kg/day)^{-1}$ derived by EPA is the geometric mean of the four estimates. This cancer potency factor is a 95% upper limit estimate; the true value is unlikely to be higher, but may be lower than this estimate. Expressed in terms of relative potency, trichloroethylene ranks in the lowest quartile among the suspect or known human carcinogens evaluated by EPA's Carcinogen Assessment Group (EPA, 1985a).

EPA (1985a) used the oral cancer potency factor to derive a potency factor of 1.3×10^{-2} $(mg/kg/day)^{-1}$ for inhalation exposure. More recently, EPA (1987b) presented an updated potency estimate of 1.7×10^{-2} $(mg/kg/day)^{-1}$ for inhalation exposure based on four sets of mouse lung tumor incidence data from two inhalation bioassays (Fukuda et al., 1983; Maltoni et al., 1986). This updated estimate is currently under review at EPA, and the earlier estimate is still listed in EPA's Integrated Risk Information (IRIS) database.

A drinking water maximum contaminant level (MCL) of 5 μg/liter and a maximum contaminant level goal (MCLG) of 0 μg/liter have been promulgated (EPA, 1987b).

The Office of Drinking Water (EPA, 1987b) issued a draft lifetime health advisory for the noncarcinogenic effects of trichloroethylene. A relative source contribution factor was not included. The advisory of 260 μg/liter was based on a study by Kimmerle and Eben (1973) that reported increased liver weights when rats were administered 55 ppm trichloroethylene for 14 weeks. Recently (EPA, 1989) also developed an oral RfD of 7.35×10^{-3} mg/kg/day for trichloroethylene based on the same study.

SUMMARY OF TRICHLOROETHYLENE CRITERIA

EPA classification	Group B2
Oral carcinogenic potency factor	1.1×10^{-2} $(mg/kg/day)^{-1}$
Inhalation carcinogenic potency factor	1.3×10^{-2} $(mg/kg/day)^{-1}$
Oral RfD	7.35×10^{-3} mg/kg/day
MCL	5 μg/liter
MCLG	0 μg/liter
Lifetime HA	260 μg/liter

REFERENCES

AGENCY FOR TOXIC SUBSTANCES and DISEASE REGISTRY (ATSDR). 1988. Draft Toxicological Profile for Trichloroethylene. U.S. Public Health Service. January 1988

ATKINSON, R. 1985. Kinetics and mechanisms of the gas-phase reactions of hydroxyl radical with organic compounds under atmospheric conditions. Chem Rev; 85:129-132 (as cited in ATSDR, 1988)

AXELSON, O. 1986. Epidemiological studies of workers with exposure to tri - and tetrachloroethylenes. Toxicol. Lett.; 31 (Suppl.):17 (As cited in ATSDR, 1988)

BADEN, J.M., KELLEY, M., MAZZE, R.I., and SIMMON, V.F. 1979. Mutagenicity of inhalation anesthetics: Trichloroethylene, divinyl ether, nitrous oxide, and cyclopropane. Br. J. Anesth.; 51:417-421 (As cited in ATSDR, 1988)

BARRET, L., FAURE, J., and DANEL, V. 1985. Epidemiological study of cancer in a community of workers occupationally exposed to trichloroethylene and cutting oils. J. Toxicol. Clin. Toxicol.; 23:4-6 (As cited in ATSDR, 1988)

BLAIR, A., DECOUFLE, P., and GRAUMAN, D. 1979. Causes of death among laundry and dry cleaning workers. Am. J. Pub. Health; 69:508-511 (As cited in ATSDR, 1988)

BRODZINSKY, R. and SINGH, G.B. 1982. Volatile organic chemicals in the atmosphere: An assessment of available data. Menlo Park, CA: Atmospheric Science Center, SRI International. Contract 68-02-3452; pp. 107-108 (As cited in ATSDR, 1988)

BRONZETTI, G., ZEIGER, E., and FREZZA, D. 1978. Genetic activity of trichloroethylene in yeast. J. Environ. Pathol. Toxicol.; 1:411-418 (As cited in ATSDR, 1988)

CALLEN, D.F., WOLF, C.R., and PHILPOT, R.M. 1980. Cytochrome P-450-mediated genetic activity and cytotoxicity of seven halogenated aliphatic hydrocarbons in Saccharomyces cerevisiae. Mutat. Res.; 77:55-63 (As cited in ATSDR, 1988)

CREBELLI, R., CONTI, G., CONTI. L., and CAURERE, A., 1985. Mutagenicity of trichloroethylene, trichloroethanol, and chloral hydrate in Aspergillis Nidulans. Mutat. Res.; 155:105-111 (As cited in ATSDR, 1988)

DILLING, W.L. 1977. Interphase transfer processes. II. Evaporation rates of chloromethanes, ethanes, ethylenes, propanes, and propylenes from dilute aqueous solutions. Comparisons with theoretical predictions. Environ. Sci. Technol. 11:405-409 (as cited in EPA, 1979)

DORFMUELLER, M.A., HENNE, S.P., YORK, R.G., BORNSCHEIN, R.L., and MANSON, J.M. 1979. Evaluation of teratogenicity and behavioral toxicity with inhalation exposure of maternal rats to trichloroethylene. Toxicology; 14:153-166 (As cited in ATSDR, 1988)

EISENREICH, S.J., LOONEY, B.B., and THORNTON, J.D., 1981. Airborne organic contaminants in the Great Lakes ecosystem. Environ Sci Technol; 15:30-8 (As cited in ATSDR, 1988)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Washington, D.C. December 1979. EPA 440/4-79-029

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Trichloroethylene. Office of Water Planning and Standards. Washington, D.C. EPA 440/5-80-077

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Trichloroethylene. Environmental Criteria and Assessment Office. Cincinnati, Ohio. EPA 540/1-86-046

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Health Assessment Document for Trichloroethylene. Environmental Criteria and Assessment Office. Research Triangle Park, N.C. EPA 600/8-82/006F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. National primary drinking water regulations; volatile synthetic organic chemicals; final rule. Fed. Reg. 50:46880-46901. (November 13, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Superfund Public Health Evaluation Manual. Office of Solid Waste and Emergency Response, Washington, D.C. October 1986. EPA/540/1-86/060

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. National primary drinking water regulations—synthetic organic chemicals; monitoring for unregulated contaminants; final rule. Fed. Reg. 52:25690-25717 (July 8, 1987)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987a. Addendum to the Health Assessment Document for Trichloroethylene: Update Carcinogenicity Assessment for Trichloroethylene. Review draft. June 1987. EPA 600/8-82/006FA

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987b. Draft Health Advisory for Trichloroethylene. Office of Drinking Water. Washington, D.C. March 31, 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, Ohio.

FUJITA, H., KOIZUMI, A., YAMAMOTO, M., KUMAI, M., SADAMOTO, T., and IKEDA, M., 1984. Inhibition of delta-aminolevulinate dehydratase in trichloroethylene-exposed rates, and the effects on heme regulation. Biochem. Biophys. Acta; 800:1-10 (As cited in ATSDR, 1988)

FUKUDA, K., TAKEMOTO, K., and TSURUTA, H. 1983. Inhalation carcinogenicity of trichloroethylene in mice and rats. Ind. Health; 21:243-254 (As cited in ATSDR, 1988)

GRANDJEAN, E., MUCHINGER, R., TURRIAN, V., HAAS, P.A., KNOWPFEL, H.K., and ROSENMUND, H. 1955. Investigations into the effect of exposure to trichloroethylene in mechanical engineering. Br. J. Ind. Med.; 12:131-142.

GREIM, H., BONSE, G., RADWAN, Z., REICHERT, D., and HENSCHLER, D. 1975. Mutagenicity in vitro and potential carcinogenicity of chlorinated ethylenes as a function of metabolic oxirane formation. Biochem. Pharmacol.; 24:2013-2017 (As cited in ATSDR, 1988)

GU, Z.W., SELE, B., JALBERT, P., et al. 1981. Induction d'echanges entre les chromatides sours (SCE) par le trichloroethylene at ses metabolites. Toxicolo. Eur. Res; 3:63-67 (As cited in ATSDR, 1988)

HARDIN, B.D., BOND, G.P., SIKOV, M.R., ANDREW, F.D., BELILES, R.P., and NIEMIER, R.W. 1981. Testing of selected workplace chemicals for teratogenic potential. Scan. J. Work Environ. Health; 7(Suppl. 4):66-75 (As cited in ATSDR, 1988)

HANSCH C. and LEO A.J., 1985. MedChem Project, Issue No. 26, Claremont, CA: Pamona College (As cited in ATSDR, 1988)

HEALY, T.E., POOLE, T.R., and HOOPER, A. 1982. Rat fetal development and maternal exposure to trichloroethylene at 100 ppm. Br J Anaesth; 54:337-341 (As cited in ATSDR, 1988)

HSDB (Hazardous Substances Data Bank). 1987. September 1987. On-line.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1982. IARC Monographs on Evaluation of the Carcinogenic Risk of Chemicals to Humans. Supplement 4. October 1982.

KAWASAKI M., 1980. Experiences with the test scheme under the chemical control law of Japan: an approach to structure-activity correlations. Ecotox Environ Safety; 4:444-54 (As cited in ATSDR, 1988)

KIMMERLE, G. and EBEN, A. 1973. Metabolism, excretion, and toxicology of trichloroethylene after inhalation. 1. Experimental exposures on rats. Arch. Toxicol.; 30:115-126 (As cited in ATSDR, 1988)

KJELLSTRAND, P., KANJE, M., MANSSON, L., BJERKAMO, M., MORTENSEN, I., LANKE, J., and HOLMQUIST, B. 1981. Trichloroethylene: Effects on body and organ weights in mice, rats, and gerbils. Toxicology; 21:105-115 (As cited in ATSDR, 1988)

KJELLSTRAND, P., HOLMQUIST, B., ALM, P., KANJE, M., ROMARE, S., JONSSON, I., MANNSON, L., and BJERKEMO, M. 1983. Trichloroethylene: further studies of the effects on body and organ weights and plasma butyl cholinesterase activity in mice. Acta. Pharmacol. Toxicol. 53:375-384 (As cited in EPA, 1985a)

KLEOPFER, R.D., EASLEY, D.M., HAAS, Jr., B.B., DEIHL, T.G., JACKSON, D.E., and WURREY, C.J. 1985. Anaerobic degradation of trichloroethylene in soil. 19(3) :277-280

LAND, P.C., OWNE, E.L., and LINDE, H.W. 1979. Mouse sperm morphology following exposure to anesthetics during early spermatogenesis. Anesthesiology; 51:S259 (Abstract). (As cited in ATSDR, 1988)

MACKAY, D. and SHIU, W.Y. 1981. A critical review of Henry's law constants for chemicals of environmental interest. J. Phys. Chem. Ref. Data; 19:1175-1179

MALTONI, C., LEFEMINE, G., and COTTI, G. 1986. Experimental research on trichloreothylene carcinogenesis. Archiv. Res. Industrial Carcinogenesis Series. Maltoni, C., Mehlman, M.A., eds. Vol. V., Princeton Scientific Publishing Co., Inc., Princeton, NJ; p 393 (As cited in ATSDR, 1988)

MCNEILL, W.C., Jr. 1979. Trichloroethylene. In: Kirk-Othmer Encyclopedia of Chemical Technology. 3rd Ed. Vol. 5. Grayson M. and Eckroth P., eds. New York: John Wiley and Sons, Inc. p. 745-753 (As cited in EPA, 1979)

NATIONAL CANCER INSTITUTE (NCI). 1976. Carcinogenesis Bioassay of Trichloroethylene. CAS No. 79-01-6. NCI-CG-TR-2.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1984. Carcinogenesis Studies of Trichloroethylene (Without Epichlorohydrin), CAS No. 79-01-6, in F344/N rats and B6C3F_1 mice (Gavage Studies). Draft. August 1983. NTP 81-84, NTP TR 243.

NATIONAL TOXICOLOGY PROGRAM (NTP) 1987. Carcinogenesis bioassay of trichloroethylene in four strains of rats. NTP TR 273. U.S. Department of Health and Human Services, National Institutes of Health, Bethesda, MD (As cited in ATSDR, 1988)

NCI (NATIONAL CANCER INSTITUTE). 1976. Carcinogenesis Bioassay of Trichloroethylene. Case No. 79-01-6, NCI-CG-TR-2, DHEW Publ. No. (NIH) 76-802 (As cited in ATSDR, 1988)

NELSON, J.L. and BULL, R.J., 1986. Induction of DNA strand breaks in rat liver with trichloroethylene (TCE). Pharmacologist; 28:182 (As cited in ATSDR, 1988)

NOMIYAMA, K., and NOMIYAMA, H. 1977. Dose-response relationship for trichloroethylene in man. Int. Arch. Occup. Environ. Health; 39:237-248.

NOMIYAMA, K., NOMIYAMA, H., and ARAI, H. 1986. Reevaluation of subchronic toxicity of trichloroethylene. Toxicol. Lett.; 31 (Suppl.):225 (Abstract) (As cited in ATSDR, 1988)

PEROCCO, P. and PRODI, G., 1981. DNA damage by haloalkanes in human lymphocytes cultured in vitro. Cancer Lett.; 13:213-218 (As cited in ATSDR, 1988)

PRICE, P.J., HASSETT, C.M., and MANSFIELD, J.L. 1978. Transforming activities of trichloroethylene and proposed industrial alternatives. In vitro, 14:290-3 (As cited in ATSDR, 1988)

PROUT, M.S., PROVAN, W.M., and GREEN, T. 1985. Species differences in response to trichloroethylene. Toxicol. Appl. Pharmacol; 79:389-400 (As sited in ATSDR, 1987)

SATO, A. and NAKAJIMA, T. 1978. Differences following skin or inhalation exposure in the absorption and excretion kintetics of trichloroethylene and toluene. Br. J. Ind. Med.; 35:43-49 (As cited in ATSDR, 1988)

SAVOLAINEN, H., PFAFFLI, P., TENGEN, M., and VAINIO, H. 1977. Trichloroethylene and 1,1,1-trichloroethane: effects on brain and liver after five days intermittent inhalation. Arch. Toxicol.; 38:229-237 (As cited in ATSDR, 1988)

SCHWETZ, B.A., LEONG, K.J., and GEHRING, P.J. 1975. The effect of maternally inhaled trichloroethylene, perchloroethylene, methylchloroform, and methylene chloride on embryonal and fetal development in mice and rats. Toxicol. Appl. Pharmacol.; 32:84-96 (As cited in ATSDR, 1988)

STOTT, N.T., QUAST, J.F., and WATANABE, P.G. 1982. Pharmacokinetics and macromolecular interactions of trichloroethylene in mice and rats. Toxicol. Appl. Pharmacol.; 62:137-151 (As cited in ATSDR, 1988)

TOLA, S., VILHUMEN, R., JARVINEN, E., and KORKOLA, M.L. 1980. A cohort study on workers exposed to trichloroethylene. J. Occup. Med.; 22:737-740 (As cited in ATSDR, 1988).

TSURUTA, H. 1978. Percutaneous absorption of trichloroethylene in mice. Ind. Health; 16:145 (As cited in ATSDR, 1988)

TU, S., MURRAY, T.A., HATCH, K.M., SIVAK, A., and MILMAN, H.A. 1985. In vitro transformation of BALB/C-3T3 cells by chlorinated ethenes and ethylenes. Cancer Lett.; 28:85-92 (As cited in ATSDR, 1988)

TUCKER, A.N., SANDERS, V.M., BARNES, D.W., et al. 1982. Toxicology of trichloroethylene in the mouse. Toxicol. Appl. Pharmacol; 62:351-357 (As cited in ATSDR, 1988)

VERSCHUEREN, K. 1983. Handbook of Environmental Data on Organic Chemicals. Van Nostrand Reinhold Co., New York

WALLES, S.A.S. 1986. Induction of single-strand breaks in DNA of mice by trichloroethylene and tetrachloroethylene. Toxicol. Lett.; 31:31-35 (As cited in ATSDR, 1988)

WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

WILSON, J.T., ENFIELD, C.G., DUNLAP, W.J., COSBY, R.L., FOSTER, D.A., and BASKIN, L.B. 1981. Transport and fate of selected organic pollutants in a sandy soil. J. Environ. Qual; 10(4): 501-506

A.10 VINYL CHLORIDE

INTRODUCTION

Vinyl chloride and polyvinyl chloride (PVC) are synthetic chemicals used as starting materials in the rubber, paper, glass, and automotive industries. They are used in the manufacture of a wide variety of products, including electrical wire insulation, piping, food packaging materials, medical supplies, and building and construction materials.

Vinyl chloride does not occur widely in the environment because of its limited release and rapid degradation (EPA, 1987a). It has not normally been detected in U.S. ambient air. Air concentrations in areas near vinyl chloride or polyvinyl chloride manufacturing facilities range from trace levels to approximately 100 $\mu g/m^3$ (ATSDR, 1988). Vinyl chloride has been detected as a relatively rare contaminant in water supplies. Concentrations up to 10 μg/liter have been reported in drinking water and surface water and levels up to 380 μg/liter have been reported in groundwater (Dyksen and Hess, 1982).

TOXICOKINETICS

Vinyl chloride is rapidly absorbed in rats following ingestion or inhalation (Bolt et al., 1977; Withey, 1976). Dermal absorption of vinyl chloride is minor (Hefner et al., 1975a). Absorbed vinyl chloride is distributed primarily to the liver and kidney, and secondarily to muscle, lung, fat, spleen, and brain (Bolt et al., 1976). The toxicity of vinyl chloride appears to be due to its metabolism in the liver to reactive polar metabolites. These metabolic processes appear to saturate at concentrations between 105 and 220 ppm in air (Hefner et al., 1975b). At low doses of vinyl chloride, metabolites are excreted primarily in the urine. At high doses most of the solvent is expired as unchanged vinyl chloride (Green and Hathaway, 1975).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Several reviews of the health effects associated with vinyl chloride exposure are available (ATSDR, 1988; EPA, 1984, 1985b; IARC, 1979).

CARCINOGENIC POTENTIAL

IARC (1979) reviewed several epidemiological studies demonstrating an association between occupational exposure to vinyl chloride and human cancer. Principally, an increased incidence of a relatively rare form of liver cancer, hepatic angiosarcomas, has been observed. A few studies have also suggested that vinyl chloride exposure may be associated with tumors at other sites including the brain, lung, lymphatic and hematopoietic systems (Bryen et al., 1976; Waxweiler et al., 1976).

Animal studies in several species support the findings of the epidemiological studies. Chronic inhalation or ingestion of vinyl chloride has been reported to induce liver tumors (liver angiosarcomas and hepatocellular carcinomas), extrahepatic angiosarcomas, Zymbal gland carcinomas, mammary gland carcinomas, brain neuroblastomas and kidney nephroblastomas in rats and/or in mice (Maltoni et al., 1981). Other studies have also reported increased tumor incidences in several target organs including the liver, Zymbal gland and nasal cavity in rats (Hong et al., 1981; Feron et al., 1979a, 1979b, 1981; Lee et al., 1978) and the liver and lung in mice (Lee et al., 1978).

GENOTOXIC POTENTIAL

The mutagenic effects of vinyl chloride have been demonstrated in metabolically activated systems using S. typhimurium, E. coli, yeast, Drosophila, and Chinese hamster V79 cells (EPA, 1985a). Chromosome aberrations have been observed in mammalian cells treated with vinyl chloride in several in vivo and in vitro assay systems and have been observed at elevated frequencies in the peripheral lymphocytes of occupationally-exposed

workers; ATSDR (1988) reviewed several of these studies. Anderson et al. (1980) reported an increase in the frequency of chromosomal aberrations in lymphocytes in workers exposed to air concentrations of approximately 50 ppm. Hansteen et al. (1978) reported a similar observation in workers exposed to air concentrations of approximately 25 ppm.

REPRODUCTIVE EFFECTS

In one epidemiological study, the incidence of fetal loss was reported to be significantly elevated among the wives of vinyl chloride workers (Infante et al., 1976; Waxweiler et al., 1977); however, studies of human populations residing near polyvinyl chloride production plants generally have not reported increased birth defect rates attributable to vinyl chloride exposure (Edmonds et al., 1975, 1978; Theriault et al., 1983).

Fetotoxic effects, including increased numbers of resorptions and decreased fetal body weight, and delayed skeletal ossification have been reported in the offspring of mice exposed to 500 ppm in air (John et al., 1977). This concentration also produced signs of maternal toxicity including reduced body weight and increased mortality. Fetotoxic effects have also been observed in rats exposed to vinyl chloride on days 1-9 of gestation, but not in rats exposed on days 8-14 or 14-21 (Ungvary et al., 1978).

Little information is available concerning the effects of vinyl chloride on the adult reproductive system. Reduced testicular weight and histopathological signs of testicular degeneration were observed in rats exposed to 100 ppm in air for up to one year (Bi et al., 1985).

ACUTE/CHRONIC EFFECTS

The liver and central nervous system are the primary target organs for vinyl chloride toxicity.

At high air concentrations (8,000-20,000 ppm), workers have experienced dizziness, headaches, euphoria, and narcosis (Nicholson et al., 1975; Lester et al., 1963). In rats, acute inhalation exposure to 50,000-150,000 ppm of vinyl chloride has been observed to induce moderate intoxication at the low dose level and deep anesthesia at the high dose level (Lester et al., 1963).

Hepatotoxic effects have been reported in occupationally-exposed workers and in experimental animals following acute or chronic inhalation or oral exposure. EPA (1985a) reviewed several of these studies. Short-term inhalation exposures to 1,000 ppm for 5-9 days in mice (Lee et al., 1977) or 5,000 ppm for 4 weeks in rats (Feron et al., 1979a) have been shown to lead to hepatotoxic effects. Similar effects have been noted in rats following longer-term exposures to 100 ppm in air for 6 months (Bi et al., 1985) or 1.3 mg/kg/day in the diet for a lifetime (Til et al., 1983).

Chronic occupational exposure to vinyl chloride has also been associated with the occurrence of "vinyl chloride disease." This syndrome is characterized by hepatotoxicity, acroosteolysis (dissolution of the ends of the distal phalanges of the hands), circulatory disturbances, scleroderma, pulmonary insufficiency and hematologic effects. Chronic studies in animals have not reproduced this syndrome (ATSDR, 1988).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

Applying EPA's criteria for evaluating the overall weight of evidence of carcinogenicity to humans, vinyl chloride has been classified in Group A–Human Carcinogen (EPA, 1984). The International Agency for Research on Cancer (IARC) has also classified vinyl chloride as a human carcinogen (IARC, 1979). EPA's Integrated Risk Information System (IRIS) does not provide information for vinyl chloride.

EPA (1984) first reported carcinogenic potency factors for exposure by inhalation and ingestion to vinyl chloride. The inhalation cancer potency factor was based on a preliminary report of an inhalation bioassay in rats

(Maltoni and Lefemine, 1975). Groups of 64 to 96 Sprague-Dawley rats were exposed to various concentrations of vinyl chloride for 4 hours per day, 5 days per week, for 52 weeks, and the survivors were sacrificed after 135 weeks. Angiosarcomas, primarily of the liver, were the predominant tumors observed; however, the incidences of male and female rats with any type of malignant tumor (6/58, 10/59, 16/69, 22/59, and 32/59 in the 0, 50, 250, 500, and 2,500 ppm dose groups, respectively) were used to calculate the cancer potency factor. The 6,000 and 10,000 ppm groups were not included in the calculation because the tumor incidence was said to have effectively plateaued at 51.7% and 62.3%. Using these data, a cancer potency factor of 2.5×10^{-2} $(mg/kg/day)^{-1}$ was calculated. More recently, this cancer potency factor has been reevaluated by the EPA on the basis of the final results of the study (Maltoni et al., 1980, 1981). The incidences of liver angiosarcomas in male and female rats exposed to air concentrations up to 30,000 ppm were used to derive a revised cancer potency value of 2.95×10^{-1} $(mg/kg/day)^{-1}$ (EPA, 1985b). This value is a 95% upper limit; the actual potency factor is unlikely to be higher, but may be lower.

The cancer potency factor for oral exposure to vinyl chloride is based on a long-term ingestion study in rats (Feron et al., 1981). Groups of male and female Wistar rats were exposed to vinyl chloride via ingestion of polyvinyl chloride powder containing some unreacted monomer. The doses of vinyl chloride administered were 0, 1.8, 5.6, and 17.0 mg/kg/day. Dosing was continued for the animals' lifetimes with terminal sacrifices at 135 weeks for males and at 144 weeks for females. Significant dose-related increases in the incidences of hepatocellular carcinomas and hepatic angiosarcomas were observed in both males and females, with angiosarcomas becoming more prevalent with increasing doses. In addition to liver tumors, increased incidences of Zymbal gland tumors, lung angiosarcomas and angiosarcomas in other extrahepatic tissues were reported. The incidences of animals with lung and/or liver tumors were used to calculate an oral cancer potency factor of 2.3 $(mg/kg/day)^{-1}$. This value is a 95% upper limit; the actual potency factor is unlikely to be higher, but may be lower.

The EPA Carcinogen Assessment Group (CAG) is presently reassessing this cancer risk estimate by evaluating the more recent data reported by Til et al. (1983) which is an extension of the earlier Feron et al. (1981) work, but includes lower doses (EPA, 1987a).

EPA promulgated a drinking water maximum contaminant level goal (MCLG) of zero because vinyl chloride is a human carcinogen. A drinking water maximum contaminant level (MCL) of 0.002 mg/liter has also been established (EPA, 1987b).

SUMMARY OF VINYL CHLORIDE CRITERIA

EPA carcinogen classification	Group A
Oral carcinogenic potency factor	2.3 $(mg/kg/day)^{-1}$
Inhalation carcinogenic potency factor	2.95×10^{-1} $(mg/kg/day)^{-1}$
Final MCL	2 μg/liter
MCLG	0 μg/liter

REFERENCES

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1988. Draft Toxicological Profile for Vinyl Chloride

ANDERSON, D., RICHARDSON, C.R., WEIGHT, T.M., and ADAMS, W.G. 1980. Chromosomal analyses in vinyl chloride exposed workers. Results from analysis 18 and 42 months after an initial sampling. Mutat. Res.; 79:151-162

BI, W., WANG, Y., HUANG, M., and MENG, D. 1985. Effect of vinyl chloride on testis in rats. Ecotox. Environ. Safety; 10(2):281-289

BOLT, H.M., KAPPUS, H., BUCHTER, A., and BOLT, W. 1976. Disposition of $(1,2^{14}C)$ vinyl chloride in the rat. Arch. Toxicol.; 35:153-162. (Cited in EPA, 1985b)

BOLT, H.M., LAIB, R.J., KAPPUS, H., and BUCHTER, A. 1977. Pharmacokinetics of vinyl chloride in the rat. Toxicology; 7(2):179-188

BRYEN, D., ENGHOLM G., ENGLUND, A., and WESTERHOLM, P. 1976. Mortality and cancer morbidity in a group of Swedish VCM and PCV production workers. Environ. Health Perspect.; 17:167-170. (Cited in EPA, 1985b)

COWFER, J.A. and MAGISTRANO, A.J. 1983. Vinyl chloride. In: Kirk-Othmer Encyclopedia of Chemical Technology. Vol. 23. Wiley Interscience, New York, NY; pp. 865-885 (As cited in ATSDR, 1988)

DYKSEN, J.E. and HESS, A.F., III. 1982. Alternatives for controlling organics in groundwater supplies. J. Am. Water Works Assoc.; 74:394-403

EDMONDS, L.D., ANDERSON, C.E., FLYNT, J.W., and JAMES, L.M. 1978. Congenital central nervous system malformations and vinyl chloride monomer exposure: A community study. Teratology; 17:137-142

EDMONDS, L.D., FALK, H., and NISSIM, J.E. 1975. Congenital malformations and vinyl chloride. Lancet; ii:1098

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Vinyl Chloride. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-078

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Vinyl Chloride. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-036

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Drinking Water Criteria Document on Vinyl Chloride. Office of Drinking Water. Washington, D.C. January 1985. Final Draft

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. National primary drinking water regulations; volatile synthetic organic chemicals, final rule. Fed. Reg. 50:46880-46901 (November 13, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Superfund Public Health Evaluation Manual. Office of Emergency and Remedial Response. EPA/540/1-86-060

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987a. Health Advisory for Vinyl Chloride. Office of Drinking Water, Criteria and Standards Division. Washington, D.C. March 31, 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987b. National primary drinking water regulations—synthetic organic chemicals; monitoring for unregulated contaminants; final rule. Fed. Reg. 52:25690-25717 (July 8, 1987)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987c. Updated Reference Dose and Cancer Potency Numbers for Use in Risk Assessments. Memorandum from Sandra Lee, Toxics Integration Branch, Washington D.C. November 16, 1987

FERON, V.J., HENDRIKSEN, C.F.M., SPEEK, A.J., TIL, H.P., and SPIT, B.J. 1981. Lifespan oral toxicity study of vinyl chloride in rats. Food Cosmet. Toxicol. 19:317-333

FERON, V.J., KRUYSSE, A., and TIL, H.P. 1979b. One-year time-sequence inhalation toxicity study of vinyl chloride in rats. I. Growth, mortality, haematology, clinical chemistry, and organ weights. Toxicol; 13:25-35

FERON, V.J., SPIT, B.J., IMMEL, H.R., and KROES, R. 1979a. One-year time-sequence inhalation toxicity study of vinyl chloride in rats. III. Morphological changes in the liver. Toxicol; 13:143-154

GREEN, T. and HATHWAY, D.E. 1975. The biological fate in rats of vinyl chloride in relation to its oncogenicity. Chem. Biol. Interact; 11:545-562

HANSTEEN, I.L., HILLESTAD, L., THIIS-EVENSEN, E., and HELDAAS, S.S. 1978. Effects of vinyl chloride in man: A cytogenetic follow-up study. Mutat. Res.; 51:271-278

HEFNER, R.E., Jr., WATANABE, P.G., and GEHRING, P.J. 1975a. Percutaneous absorption of vinyl chloride: Toxicol. Appl. Pharmacol.; 34:529-532

HEFNER, R.E., Jr., WATANABE, P.G., and GEHRING, P.J. 1975b. Preliminary studies of the fate of inhaled vinyl chloride monomer in rats. Ann NY Acad. Sci.; 246:135-148

HILL, J.N., KOLLIG, H.P., PARIS, D.F., WOLFE, N.L., and ZEPP, R.G. 1976. Dynamic behavior of vinyl chloride in aquatic ecosystems. Environmental Protection Agency. Office of Research and Development, Athens, Georgia. EPA 60013-76-001 (As cited in EPA, 1979)

HONG, C.B., WINSTON, J.M., THORNBURG, L.P., and LEE, C.C. 1981. Follow-up study on the carcinogenicity of vinyl chloride and vinylidene chloride in rats and mice: Tumor incidence and mortality subsequent to exposure. J. Toxicol. Environ. Health; 7:909-924

INFANTE, P.F., WAGONER, J.K., McMICHAEL, A.J., WAXWEILER, R.J., and FALK, H. 1976. Genetic risks of vinyl chloride. Lancet (April 3); 734-735

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1979. IARC Monographs on the Evaluation of the Carcinogenic Risks of Chemicals to Humans. Vol. 20: Some Halogenated Hydrocarbons. World Health Organization, Lyon, France

JOHN, J.A., SMITH, F.A., LEONG, B.K.J., and SCHWETZ, B.A. 1977. The effects of maternally inhaled vinyl chloride on embryonal and fetal development in mice, rats and rabbits. Toxicol. Appl. Pharmacol.; 39:497-513

LEE, C.C., BHANDARI, J.C., and WINSTON, J.M. 1977. Inhalation toxicity of vinyl chloride and vinylidene chloride. Environ. Health Perspect.; 21:25-32

LEE, C.C., BHANDARI, J.C., WINSTON, J.M., HOUSE, W.B., DIXON, R.L., and WOODS, J.S. 1978. Carcinogenicity of vinyl chloride and vinylidene chloride. J. Toxicol. Environ. Health; 4:15-30

LESTER, D., GREENBERG, L.A., and ADAMS, W.R. 1963. Effects of single and repeated exposures of humans and rats to vinyl chloride. Am. Ind. Hyg. Assoc. J.; 24:265-275

MALTONI, C. and LEFEMINE, G. 1975. Carcinogenicity bioassays vinyl chloride. Current results. Ann. NY Acad. Sci. 246:195-218

MALTONI, C., LEFEMINE, G., CILIBERTI, A. et al. 1980. Vinyl chloride carcinogenicity bioassays (BT project) as an experimental model for risk identification and assessment in environmental and occupational carcinogenesis. Epidemiol Anim Epidemiol Hum: Cas Chlorure Vinyle Monomere (Reun Club Cancerog Chim), 20th, Meeting Date 1979, pp. 11-112. Publ Essent, Paris, France

MALTONI, C., LEFEMINE, G., CILIBERTI, A. et al. 1981. Carcinogenicity bioassays of vinyl chloride monomer: A model of risk assessment on an experimental basis. Environ. Health Perspect.; 41:329

NICHOLSON, W.J., HAMMOND, E.C., SEIDMAN, H., and SELIKOFF, I.J. 1975. Mortality experience of a cohort of vinyl chloride-polyvinyl chloride workers. Ann. NY Acad. Sci.; 246:225-230

THERIAULT, G., ITURRA, H., and GINGRAS, S. 1983. Evaluation of the association between birth defects and exposure to ambient vinyl chloride. Teratology; 27:359-370

TIL, H.P., IMMEL, H.R., and FERON, V.J. 1983. Lifespan Oral Carcinogenicity Study of Vinyl Chloride in Rats. Final Report. Civo Institutes TNO Report No. V 83 285/29109

UNGVARY, G., HUDAK, A., TATRAI, E., LORINCZ, M., and FOLLY, G. 1978. Effects of vinyl chloride exposure alone and in combination with trypan blue applied systematically during all thirds of pregnancy on the fetuses of CFY rats. Toxicology; 11(1):45-54

VERSCHUEREN, K. 1983. Handbook of Environmental Data on Organic Chemicals. Van Nostrand Reinhold Co., New York. 659 pages

WAXWEILER, R.J., STRINGER, W., WAGNER, J.K., JONES, J., FALK, H., and CARTER, C. 1976. Neoplastic risk among workers exposed to vinyl chloride. Ann NY Acad Sci; 271:40-48

WAXWEILER, R.J., FALK, H., McMICHAEL, A., MALLOV, J.S., and GRIVAS, A.S. 1977. A cross-sectional epidemiologic survey of vinyl chloride workers. NTIS PB274193

WITHEY, J.R., 1976. Pharmacodynamics and uptake of vinyl chloride monomer administered by various routes to rats. J. Toxicol. Environ. Health; 1:381-394

APPENDIX B

ESTIMATION OF VAPOR AND PARTICULATE EMISSIONS AND ATMOSPHERIC DISPERSION AT THE WDI SITE

Appendix B

Table of Contents

List of Tables

APPENDIX B

ESTIMATION OF VAPOR AND PARTICULATE EMISSIONS AND ATMOSPHERIC DISPERSION AT THE WDI SITE

Contaminant release and air dispersion models were used to estimate potential exposures to area residents from inhalation of chemicals associated with airborne particulates and subsurface gases from the WDI site. Emissions of volatile chemicals by diffusion of subsurface gas was estimated by an equation developed by Farmer (1978). A mathematical model of wind erosion from surface soils developed by Cowherd et al. (1984) was used to estimate airborne particulate generation by wind erosion at the site. Dispersion and airborne transport of the emissions estimated by the two procedures was evaluated using a Gaussian dispersion model developed by Turner (1970). Subsurface gas infiltration rates and predicted indoor air concentrations are also discussed.

B-1 ESTIMATION OF VAPOR EMISSIONS

Emissions of vapor-phase chemicals from site soils were estimated using an equation developed by Farmer et al. (1978) to estimate volatilization through landfill covers. Farmer's equation, as modified by Shen (1981) and Farino et al. (1983), and as described in EPA (1988) is:

$$E_i = D_i \, C_{si} \, A \, (P_t^{4/3}) \, M_i / d_{sc} \qquad \text{Eq. B-1}$$

where,

- E_i = emission rate of component i of waste (g/sec),
- D_i = diffusivity of component i in air (cm^2/sec),
- C_{si} = saturation vapor concentration of component i (g/cm^3),
- A = exposed surface area (cm^2),
- P_t = total soil porosity (unitless),
- M_i = mole fraction of component i in the waste (g-mole/g-mole),
- d_{sc} = effective depth of soil cover (cm).

Since the chemical is already present in a gaseous form, M_i is equal to 1.0. The above equation was modified to reflect the use of subsurface gas concentrations instead of waste concentrations:

$$E_i = D_i \, C_{sg} \, A \, (P_t^{4/3})/d_{sc} \qquad \text{Eq. B-2}$$

where,

C_{sg} - concentration of chemical in soil gas.

All other terms are as in Equation B-1. The Farmer model assumes that migration through the soil is a steady state process in which the soil concentrations remain constant over the period of release. Thus, the equation may overestimate long-term exposures if the mass of chemical initially present in the soil is small.

Diffusivities for the organic chemicals of potential concern are shown in Table B-1. The contaminated area was assumed to be 85,200 m^2, the area of unpaved soil at the site. The total soil porosity was assumed to be 44%, the geometric mean value measured at the site. The effective depth of subsurface cover was assumed to be 1.6 meters, the average depth to the top of the subsurface gas probes. The saturated vapor concentrations were assumed to be the geometric mean soil gas concentrations of the volatile organic chemicals of potential concern.

Estimated emission rates for volatile organics present in subsurface gas at the WDI site are shown in Table B-1.

B-2 ESTIMATION OF PARTICULATE EMISSIONS

Wind erosion was evaluated using a procedure developed by Cowherd et al. (1984) for an "unlimited reservoir" of erodible particles. The procedure uses soil characteristics and wind speed to estimate the grams per second of respirable particulates (PM_{10}) emitted from the site.

TABLE B-1
ESTIMATED AMBIENT AIR CONCENTRATIONS
FOR VAPOR EMISSIONS FROM WDI SITE

COMPOUND	Diffusivity (m2/sec)	Subsurface Gas Average Case Conc. (ug/m3) (a)	Emission Rate (ug/s)	Geometric Mean Estimated Ambient Air Concentration (ug/m3) at 0.1 km	at 0.5 km	at 1 km	Subsurface Gas Plausible Maximum Case Conc. (ug/m3) (b)	Emission Rate (ug/s)	Maximum Estimated Ambient Air Concentration (ug/m3) at 0.1 km	at 0.5 km	at 1 km
Benzene	8.7E-06	99	15	6.0E-03	1.1E-03	5.9E-04	524	131	5.1E-02	9.6E-03	5.0E-03
Carbon Tetrachloride	8.0E-06	1.7	0.24	9.5E-05	1.8E-05	9.3E-06	9.4	2.2	8.4E-04	1.6E-04	8.3E-05
Chloroform	8.9E-06	3.8	0.60	2.4E-04	4.4E-05	2.3E-05	45	11	4.5E-03	8.4E-04	4.4E-04
1,2-Dibromoethane	8.6E-06	160	25	9.6E-03	1.8E-03	9.4E-04	209	52	2.0E-02	3.8E-03	2.0E-03
1,2-Dichloroethane	9.4E-06	42	7.1	2.8E-03	5.2E-04	2.7E-04	117	32	1.2E-02	2.3E-03	1.2E-03
Tetrachloroethylene	7.4E-06	88	12	4.5E-03	8.5E-04	4.4E-04	88	19	7.3E-03	1.4E-03	7.1E-04
1,1,1-Trichloroethane	7.9E-06	44	6.2	2.4E-03	4.6E-04	2.4E-04	130	29	1.2E-02	2.2E-03	1.1E-03
Trichloroethylene	8.1E-06	150	22	8.5E-03	1.6E-03	8.3E-04	215	50	2.0E-02	3.7E-03	1.9E-03
Vinyl Chloride	1.1E-05	38	7.2	2.8E-03	5.3E-04	2.8E-04	945	290	1.1E-01	2.1E-02	1.1E-02

(a) - Subsurface gas average case concentrations calculated using one half the detection limit for nondetects.
(b) - Subsurface gas plausible maximum case concentrations calculated using positive detects only.

Parameters required for the Cowherd model are: soil particle size distribution, soil roughness height, fraction of vegetative cover, average annual wind speed, and area of the source. The mode of the aggregate particle size distribution was assumed to be 0.1 mm, based on the soil types reported in surface soils at the site (silty sands and silts). A roughness height of 2 cm was used, which corresponds to a partially vegetated open area (Cowherd, 1984). A vegetative cover factor of 20% was used, which represents a weedy but not heavily vegetated terrain. The average wind speed was assumed to be 2.4 m/s, based on the meteorological data collected by EBASCO (1989).

The area of the site from which dusts may potentially be generated was assumed to be 85,200 m^2, the area of exposed soil at the site. The quantity of respirable dust generated by wind erosion was calculated by the procedure outlined in Cowherd (1984) to be 0.19 grams/second. The concentrations of contaminants in the respirable particulates (PM_{10}) were assumed to be the same as their geometric mean concentrations in surface soils at the site. Emissions calculated for the contaminants of potential concern are shown in Table B-2.

B-3 ESTIMATION OF AIR CONCENTRATIONS

Exposure point concentrations for particulate-bound chemicals and for volatile organic chemicals in subsurface gas were estimated for area residents living and students attending school downwind from the WDI facility. Concentrations were estimated for residents living at distances of 0.1 km, 0.5 km, and 1 km from the site boundary and for students at a distance of 0.1 km from the site boundary.

A Gaussian equation for atmospheric dispersion described by Turner (1970) was used to determine off-site air concentrations. The Turner equations determine the dispersion of an atmospheric plume at a specified distance downwind from an point source of atmospheric emissions. Emissions from the WDI site take place over an area source rather than from a point source such as a stack or vent; therefore, emissions were assumed to originate

TABLE B-2

ESTIMATED AMBIENT AIR CONCENTRATIONS
OF PARTICULATES EMITTED FROM SURFACE SOILS
VIA WIND EROSION

Chemical	GEOMETRIC MEAN SOIL CONC. (MG/KG)	EMISSION RATE (mg/sec)	ESTIMATED AIR CONCENTRATIONS (mg/m3)		
			at 0.1 km	at 0.5 km	at 1 km
INORGANICS					
Antimony	5.4	1.0E-03	4.0E-07	7.5E-08	3.9E-08
Arsenic	8.0	1.5E-03	6.0E-07	1.1E-07	5.8E-08
Cadmium	1.1	2.1E-04	8.1E-08	1.5E-08	7.9E-09
Chromium	23	4.4E-03	1.7E-06	3.2E-07	1.7E-07
Copper	36	6.8E-03	2.7E-06	5.0E-07	2.6E-07
Lead	43	8.2E-03	3.2E-06	6.0E-07	3.1E-07
Manganese	364	6.9E-02	2.7E-05	5.1E-06	2.6E-06
Mercury	0.15	2.8E-05	1.1E-08	2.1E-09	1.1E-09
Selenium	0.5	9.5E-05	3.7E-08	7.0E-09	3.6E-09
Thallium	7.9	1.5E-03	5.9E-07	1.1E-07	5.8E-08
ORGANICS					
Benzene	0.26	4.9E-05	1.9E-08	3.6E-09	1.9E-09
2-Butanone	0.0082	1.6E-06	6.1E-10	1.1E-10	6.0E-11
Chlordane	0.010	1.9E-06	7.4E-10	1.4E-10	7.3E-11
DDT	0.14	2.6E-05	1.0E-08	1.9E-09	9.9E-10
Dieldrin	0.038	7.2E-06	2.8E-09	5.3E-10	2.8E-10
Ethylbenzene	0.047	8.9E-06	3.5E-09	6.6E-10	3.4E-10
Heptachlor epoxide	0.010	1.8E-06	7.1E-10	1.3E-10	7.0E-11
Methylene Chloride	0.023	4.4E-06	1.7E-09	3.2E-10	1.7E-10
Pentachlorophenol	0.25	4.7E-05	1.8E-08	3.4E-09	1.8E-09
Toluene	0.052	9.9E-06	3.9E-09	7.3E-10	3.8E-10
Xylenes	0.19	3.6E-05	1.4E-08	2.7E-09	1.4E-09
PAHs					
Carcinogenic	0.89	1.7E-04	6.6E-08	1.2E-08	6.5E-09
Noncarcinogenic	4.4	8.4E-04	3.3E-07	6.2E-08	3.2E-08
PCBs	2.1	3.9E-04	1.5E-07	2.9E-08	1.5E-08

from a virtual point source upwind of the facility, as recommended by Turner (1970).

By assuming that the wind speed and emission rate remained constant during the time that it takes for vapors to travel from the site to the off-site receptors, it was possible to estimate the diffusion or dilution of emissions away from the source. A neutral (D) atmospheric stability class was used in these estimates, as suggested in the Superfund Exposure Assessment Manual (EPA, 1988) when making conservative estimates of long-term mean ambient air concentrations. The Turner model does not account for the variation in wind speed with height. In addition, the model assumes that the stability classification is the same throughout the diffusing layer and that no turbulent transfer occurs through layers which have different stabilities.

The special case of defining a ground-level area source as a virtual point source was used to calculate ambient air concentrations off-site. For this case the following dispersion equation is used:

$$X = \frac{Q\ F\ Y}{\pi\ s_y\ s_z\ U}$$

where;

- X - concentration of gas or aerosol ($\mu g/m^3$),
- Q - emission rate of erodible materials or subsurface gases (g/s),
- F - percentage of time during which the wind blows towards the exposure point (dimensionless),
- Y - conversion factor (10^6 $\mu g/g$),
- s_y - standard deviation of plume concentration distribution in the horizontal plane (m),
- s_z - standard deviation of plume concentration distribution in the vertical plane (m), and
- U - mean wind speed (m/sec).

When an area source is defined as a virtual point source, it is first necessary to calculate an initial standard deviation of the plume concentration in the horizontal, s_{yo}. For a square area source such as the WDI site, this can be approximated by $s_{yo} \approx S/4.3$, where S is the length of a side of the area source. A virtual downwind distance, x_y, can then be found by using Figure 3-2 in Turner (1970) for a stability class D. The vertical standard deviation, s_z, can be found by applying the actual downwind distance, x, to Figure 3-3 of Turner (1970). For the WDI site, the downwind distance was assumed to be the distance from the center of the site to a receptor 0.1 km, 0.5 km, or 1 km downwind. The final horizontal dispersion value, s_y, is calculated by applying the distance $x + x_y$ to Figure 3-2 of Turner (1970).

The wind speed, U, was assumed to be 2.4 m/sec, based on meteorological data collected by EBASCO (1989). The frequency of time that wind blows toward the exposure point was conservatively assumed to be 100 percent. Air concentrations for volatile organic chemicals are shown in Table B-1. The air concentrations of the semivolatile and inorganic chemicals of concern are shown in Table B-2.

B-4 CALCULATION OF SUBSURFACE GAS INFILTRATION AND EMISSION RATES

Calculation of the soil gas infiltration and emission rates were made for potential future residences on the WDI site. The infiltration was assumed to occur along the perimeter of the house's foundation. Since no carrier gas is present (i.e., methane or carbon dioxide), the driving force for the infiltration was the differential atmospheric pressure between the soil column and inside the house. This differential pressure was assumed to result in an infiltration flow rate of 1 m^3/hr for the incoming soil gas (Scott, 1983). The flow rate was divided by the area of infiltration (in m^2) to determine the infiltration velocity (in m/hr). The product of the soil gas velocity (in m/s) and the soil gas concentration (in g/m^3) is the chemical-specific flux rate into the structure.

The subsurface gas concentrations listed in Table 2-6 were used to determine the chemical-specific flux rates. The product of these flux rates and the area over which subsurface gas infiltration was assumed to occur was the emission rate listed in Table B-3. The area of infiltration was estimated by assuming that the infiltration occurred through a 3.18×10^{-3} m (1/8 inch) crack along the entire perimeter of the home. The homes were assumed to be square, with a total surface area of 139.4 m^2 (~ 1,521 ft^2). Each side of the house was assumed to be 11.8m (~39 feet) in length. The total area of infiltration is then given by:

Total area of infiltration (m^2) = $4 \times (11.81 \text{ m} \times 3.18 \times 10^{-3} \text{ m})$
= 1.50×10^{-1} m^2

The factor of 4 in the equation accounts for the fact that infiltration is assumed to occur along all four walls of the home.

Once the emission rates had been calculated, the associated indoor air concentration was estimated by using a one compartment indoor air model. This model assumes that the infiltrating gas is uniformly and instantaneously mixed within the entire compartment. The indoor air concentration is the ratio of the emission rate and the rate at which indoor air is exchanged with uncontaminated outdoor air. Thus the indoor air concentration is given by:

Indoor Air Conc. (g/m^3) = [Emission Rate (g/s)]/[Air Exchange Rate (m^3/s)]

The air exchange rate reflects the rate at which indoor air is replaced by outdoor air. It is a function of temperature and pressure differences between the indoor and outdoor air, and the tightness of the structure. In this analysis the air exchange rate is exclusive of air provided for ventilation. The air exchange rate (AER) in m^3/s can be estimated by:

[(# of air exchanges/hour)(volume of air exchanged, m^3)]/(3,600 s/hr)

For residential structures, it was assumed that the number of air exchanges per hour was 0.25. This value is representative of very tight structures and will provide an upper bound estimate of the indoor air concentrations. The volume of air exchanged is the product of the floor surface area and the height of the room. The height of the room was assumed to be 2.44 m (8 ft). Using the floor surface area given earlier, the resulting air volume exchanged was 340.1 m^3 for the home. The resulting air exchange rate was 2.36×10^{-2} m^3/s. The air exchange rates were used with the appropriate emission rates estimated for the homes and the commercial development to estimate the indoor air concentrations given in Table B-3.

TABLE B-3
EXPOSURE POINT CONCENTRATIONS OF SUBSURFACE GAS CONTAMINANTS
FOR ON-SITE RESIDENTS
WDI SITE

COMPOUND	Average Case Indoor Air Concentration (a) (ug/m3)	Plausible Maximum Case Indoor Air Concentration (b) (ug/m3)
Benzene	1.2E+00	6.3E+00
Carbon Tetrachloride	2.1E-02	1.1E-01
Chloroform	4.6E-02	5.4E-01
1,2-Dibromoethane	1.9E+00	2.5E+00
1,2-Dichloroethane	5.1E-01	1.4E+00
Tetrachloroethene	1.1E+00	1.1E+00
1,1,1-Trichloroethane	5.3E-01	1.6E+00
Trichloroethene	1.8E+00	2.6E+00
Vinyl Chloride	4.6E-01	1.1E+01

(a) Based on geometric mean of all samples.
(b) Based on geometric mean of positively detected samples only.

B-5 REFERENCES

COWHERD, C., MULESKI, G.E., ENGLEHART, PLHL, and GILLETTE, D.A. 1985. Rapid Assessment of Exposure to Particulate Emissions from Surface Contamination Sites. Kansas City, MO. Midwest Research Institute. PB85-192219.

EBASCO SERVICES, INC. (EBASCO). 1989. Subsurface Gas Characterization Report. Volume I. May 1989.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Superfund Exposure Assessment Manual. Office of Remedial Response. Washington, D.C. EPA/540/1-88-001. April.

FARINO, W., SPAWN, P. JASINSKI, M., and MURPHY, B. 1983. Evaluation and selection of models for estimating air emissions from hazardous waste treatment, storage and disposal facilities. Revised Draft Final Report. GCA Corporation. Bedford, Mass. Prepared for U.S. EPA Office of Solid Waste. Land Disposal Branch. Contract No. 68-02-3168.

FARMER, W.J., YANG, M.S., LETEY, J. SPENCER, W.F., and ROULIER, M.H. 1978. Land disposal of hexachlorobenzene waste: controlling vapor movement in soils. San Antonio, TX. 4th Annual Symposium on Land Disposal.

NATIONAL OCEANIC AND ATMOSPHERIC ADMINISTRATION (NOAA). 1983. Preparing Meteorological Data for Use in Routine Dispersion Calculations - Workgroup Summary Report. NOAA Technical Memorandum ERL ARL-122. August 1983.

SCOTT, A. 1983. Review of Existing Instrumentation and Evaluation for Research to Determine Levels of Radon at a Proposed Building Site. January 1983.

SHEN, T. 1981. Estimating hazardous air emissions from disposal sites. Pollution Engineering 13(8): 31-34.

TURNER, B. 1970. Workbook of Atmospheric Dispersion Estimates. Air Resources Field Office, Environmental Science Services Administration.

APPENDIX C

SAMPLE RISK CALCULATIONS

APPENDIX C -- SAMPLE RISK CALCULATIONS

Sample risk calculations are presented below for the direct contact, the inhalation, and groundwater ingestion pathways.

Direct Contact

The direct contact pathway was complete for trespasssers under current-use conditions and residents under potential future-use conditions. The CDI for direct contact was calculated using Equations 5-1 through 5-4. For trespassers, the exposure point concentrations are found in Table 4-6; the exposure assumptions for trespassers are in Table 5-6. For future residents, exposure point concentrations are found in Table 4-10; exposure assumptions are listed in Table 5-13.

EXAMPLE 1 DIRECT CONTACT WITH CARCINOGENS: For trespassers, carcinogenic risk associated with average case exposure to arsenic is calculated as follows:

$ITK_{dc} = ITK_i + ITK_d$ (Equation 5-1)

$ITK_i = C * BIO * IN * Y$ (Equation 5-2)

$ITK_d = C * ABS * S * SA * Y$ (Equation 5-3)

$ITK_{dc} = (C * Y) * [(BIO * IN) + (ABS * S * SA)]$

C	=	8.0 mg/kg	(from Table 4-6)
Y	=	1 kg/10^6 mg	
BIO	=	0.8	(from Table 5-6)
IN	=	100 mg	(from Table 5-6)
ABS	=	0	(from Table 5-6)
S	=	1.45 mg/cm^2/day	(from Table 5-6)
SA	=	1400 cm^2	(from Table 5-6)

$ITK_{dc} = (8.0 \text{ mg/kg} * 1 \text{ kg}/10^6 \text{ mg}) * [(0.8 * 100 \text{ mg}) + (0 * 1.45 \text{ mg/cm}^2\text{/day} * 1400 \text{ cm}^2)]$

$ITK_{dc} = 6.4 \times 10^{-4}$ mg/day

$CDI_{dc} = (ITK_{dc} * D * F) / (BW * E * 365)$ (Equation 5-4)

D - 4 years (from Table 5-6)
F - 52 days/year (from Table 5-6)
BW - 60 kg (from Table 5-6)
E - 75 years (from Table 5-6)

CDI_{dc} - (6.4 x 10^{-4} mg/day * 4 years * 52 days/year)/(60 kg * 75 years * 365 days/year)

CDI_{dc} - 8.1 * 10^{-8} mg/kg/day

Cancer Risk - CDI_{dc} * CPF

CPF - 2.0 $(mg/kg/day)^{-1}$ (from Table 3-1)

Cancer Risk - (8.1 * 10^{-8} mg/kg/day) * (2.0 $(mg/kg/day)^{-1}$)

Cancer Risk - 4 x 10^{-8}

EXAMPLE 2 DIRECT CONTACT WITH NONCARCINOGENS: Under plausible maximum conditions, noncarcinogenic risk associated with noncarcinogenic PAHs for trespassers via direct contact exposure is calculated as follows:

ITK_{dc} - ITK_i + ITK_d (Equation 5-1)
ITK_i - C * BIO * IN * Y (Equation 5-2)
ITK_d - C * ABS * S * SA * Y (Equation 5-3)

ITK_{dc} - (C * Y) * [(BIO * IN) + (ABS * S * SA)]

C - 17.9 mg/kg (from Table 4-6)
Y - 1 kg/10^6 mg
BIO - 0.5 (from Table 5-6)
IN - 100 mg (from Table 5-6)
ABS - 0.05 (from Table 5-6)
S - 2.77 mg/cm^2/day (from Table 5-6)
SA - 1980 cm^2 (from Table 5-6)

ITK_{dc} - (17.9 mg/kg *1 kg/10^6 mg) * [(0.5*100 mg) + (0.05 * 2.77 mg/cm^2/day * 1980 cm^2)]

ITK_{dc} - 5.8 x 10^{-3} mg/day

CDI_{dc} - (ITK_{dc} * D * F) / (BW * E * 365) (Equation 5-4)

D - 6 years (from Table 5-6)
F - 260 days/year (from Table 5-6)
BW - 60 kg (from Table 5-6)
E - 6 years (from Table 5-6)

CDI_{dc} = $(5.8 \times 10^{-3}$ mg/day * 6 years * 260 days/year)/(70 kg * 6 years * 365 days/year)

CDI_{dc} = $5.9 * 10^{-5}$ mg/kg/day

CDI:RfD = CDI_{dc}/RfD

RfD = 0.41 mg/kg/day (from Table 3-1)

CDI:RfD = $(5.9 * 10^{-5}$ mg/kg/day)/(0.41 mg/kg/day)

CDI:RfD = 1×10^{-4}

Inhalation

The inhalation pathway was complete for students at St. Paul's High School and off-site residents under current-use conditions and on-site residents under potential future-use conditions. The CDI for inhalation was calculated using Equations 5-5 through 5-6. As discussed in Appendix B, exposure point concentrations under current-use conditions were calculated in two ways: 1) subsurface gas concentrations were used to estimate release of volatile organic chemicals to air, and 2) surface soil concentrations were used to estimate fugitive dust emissions. The exposure point concentrations for volatile contaminants in subsurface gas are found in Table 4-7; exposure point concentrations for fugitive dust emissions are found in Table 4-8. The exposure assumptions for students and residents are in Table 5-8.

Under future-use condions, inhalation risk estimates were based on exposure to subsurface gas infiltrating into homes. Exposure point concentrations for this scenario are found in Table 4-12; Table 5-19 lists the exposure assumptions.

EXAMPLE 3 INHALATION OF CARCINOGENS IN SUBSURFACE GAS: For current residents living within 0.1 km of the WDI site, cancer risk associated with the inhalation of vinyl chloride present in subsurface gas under average case conditions was estimated as follows:

ITK_a = C * I * L * A * Y (Equation 5-5)

C - 2.8×10^{-6} mg/m^3 (from Table 4-7)
I - 20 m^3 (from Table 5-8)
L - 24 hour/day (from Table 5-8)
Y - 1 day/ 24 hours
A - 1 (from Table 5-8)

ITK_a - 2.8×10^{-6} mg/m^3 * 20 m^3 * 24 hour/day * 1 day/ 24 hours * 1

ITK_a - 5.6×10^{-5} mg/day

CDI_a - $(ITK_a * D * F) / (BW * E * 365)$

D - 9 years (from Table 5-8)
F - 330 days/year (from Table 5-8)
BW - 70 kg (from Table 5-8)
E - 75 years (from Table 5-8)

CDI_a - $(5.6 \times 10^{-5}$ mg/day * 9 years * 330 days/year)/(70 kg * 75 years * 365 days/year)

CDI_a - 8.7×10^{-8} mg/kg/day

Cancer Risk - CDI_a * CPF

CPF - 0.295 (mg/kg/day)$^{-1}$ (from Table 3-2)

Cancer Risk - 8.7×10^{-8} mg/kg/day * 0.295 (mg/kg/day)$^{-1}$

Cancer Risk - 3×10^{-8}

EXAMPLE 4 INHALATION OF NONCARCINOGENS IN PARTICULATES: Under plausible maximum conditions, noncarcinogenic risk associated with lead for students at St. Paul's High School via inhalation of airborne particulate is calculated as follows:

ITK_a - C * I * L * A * Y (Equation 5-5)

C - 3.2×10^{-6} mg/m^3 (from Table 4-8)
I - 20 m^3 (from Table 5-8)
L - 8 hour/day (from Table 5-8)
Y - 1 day/ 24 hours
A - 1 (from Table 5-8)

ITK_a - 3.2×10^{-6} mg/m^3 * 20 m^3 * 8 hour/day * 1 day/ 24 hours * 1

ITK_a - 2.1×10^{-5} mg/day

CDI_a - $(ITK_a * D * F) / (BW * E * 365)$

D	-	4 years	(from Table 5-8)
F	-	180 days/year	(from Table 5-8)
BW	-	60 kg	(from Table 5-8)
E	-	4 years	(from Table 5-8)

CDI_a - $(2.1 \times 10^{-5}$ mg/day * 4 years * 180 days/year)/(60 kg * 4 years * 365 days/year)

CDI_a - 1.7×10^{-7} mg/kg/day

CDI:RfD - CDI_a/RfD

RfD - 6×10^{-4} mg/kg/day (from Table 3-2)

CDI:RfD - $(1.7 * 10^{-7}$ mg/kg/day) / $(6 \times 10^{-4}$ mg/kg/day)

CDI:RfD - 3×10^{-4}

Groundwater Ingestion

The groundwater ingestion pathway was complete for future on-site residents under future-use conditions. The CDI for groundwater ingestion was calculated using Equations 5-7 and 5-8. The exposure point concentrations are found in Table 4-11; the exposure assumptions for are listed in Table 5-16.

EXAMPLE 5 DRINKING WATER INGESTION OF CARCINOGENS: For adult residents, carcinogenic risk associated with average case exposure to tetrachloroethylene in drinking water is calculated, as follows:

ITK_g - $C_w * W * G$ (Equation 5-7)

C_w	-	2.6 ug/liter	(from Table 4-11)
W	-	2 liter/day	(from Table 5-16)
G	-	1	(from Table 5-16)

ITK_g - 2.6 ug/liter * 2 liter/day * 1

ITK_g - 5.2×10^{-3} mg/day

CDI_g - $(ITK_g * D * F) / (BW * E * 365)$ (Equation 5-8)

D	-	9 years	(from Table 5-16)
F	-	365 days/year	(from Table 5-16)
BW	-	70 kg	(from Table 5-16)
E	-	75 years	(from Table 5-16)

CDI_g = $(5.2 \times 10^{-3}$ mg/day * 9 years * 365 days/year)/(70 kg * 75 years * 365 days/year)

CDI_g = 8.9×10^{-6} mg/kg/day

Cancer Risk = CDI_g * CPF * 2

CPF = 1×10^{-2} $(mg/kg/day)^{-1}$ (from Table 3-1)
2 = Factor to adjust for volatilization.

Cancer Risk = 8.9×10^{-6} mg/kg/day * 1×10^{-2} $(mg/kg/day)^{-1}$ * 2

Cancer Risk = 2×10^{-7}

EXAMPLE 6 DRINKING WATER INGESTION OF NONCARCINOGENS: For child residents consuming groundwater containing lead under plausible maximum conditions, the CDI was calculated as follows:

ITK_g = C_w * W * G (Equation 5-7)

C_w = 16 ug/liter (from Table 4-11)
W = 1 liter/day (from Table 5-16)
G = 1 (from Table 5-16)

ITK_g = 16 ug/liter * 1 liter/day * 1

ITK_g = 1.6×10^{-2} mg/day

CDI_g = $(ITK_g * D * F) / (BW * E * 365)$ (Equation 5-8)

D = 2 years (from Table 5-16)
F = 365 days/year (from Table 5-16)
BW = 10 kg (from Table 5-16)
E = 2 years (from Table 5-16)

CDI_g = $(1.6 \times 10^{-2}$ mg/day * 2 years * 365 days/year)/(10 kg * 2 years * 365 days/year)

CDI_g = 1.6×10^{-3} mg/kg/day

CDI:RfD = CDI_g/RfD

RfD = 6×10^{-4} mg/kg/day (from Table 3-1)

CDI:RfD = $(1.6 * 10^{-3}$ mg/kg/day) / $(6 \times 10^{-4}$ mg/kg/day)

CDI:RfD = 3